VOLUME 18 NUMBER 4

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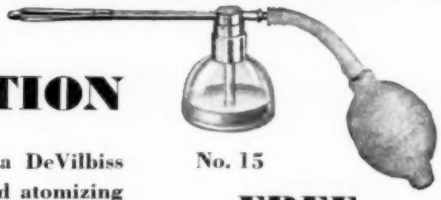

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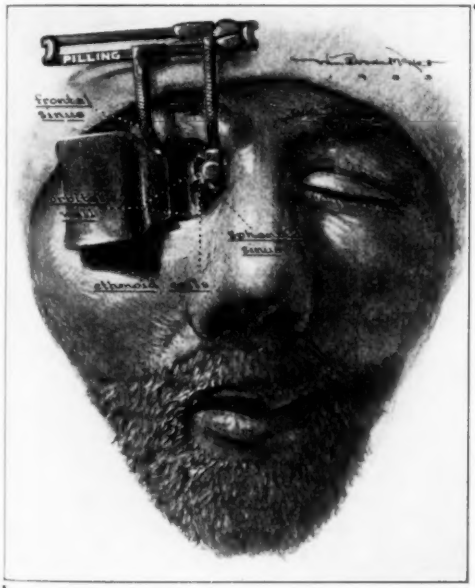

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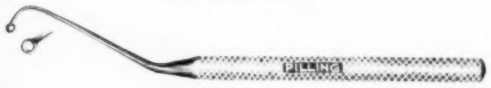

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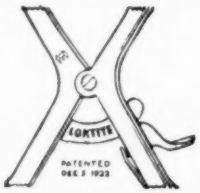

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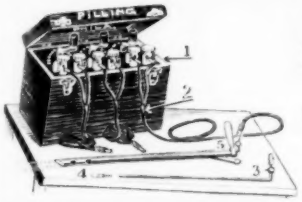

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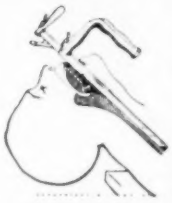

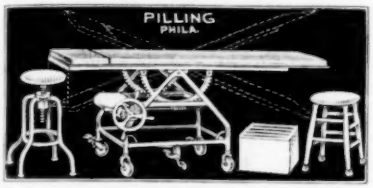

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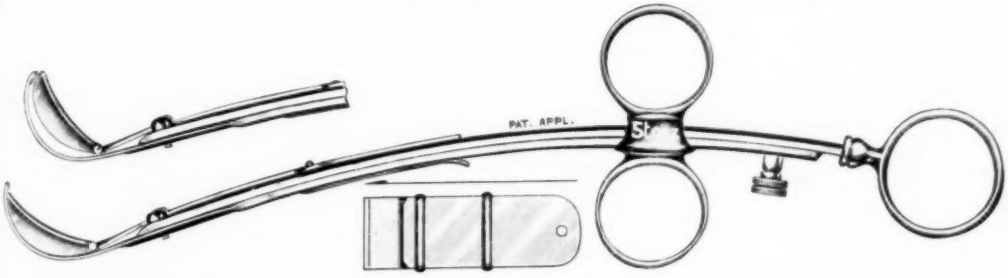

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VOLUME 18 OCTOBER, 1933 NUMBER 4

INNERVATION OF THE LARYNX

III. EXPERIMENTAL PARALYSIS OF THE LARYNGEAL NERVE

FREDERICK LEMERE, M.D.

DENVER

The first obstacle to an understanding of the problem of paralysis of the laryngeal nerves is the lack of agreement as to the innervation of the laryngeal muscles. The classic dictum of Ónodi,[1] however, may be accepted as accurate. His conclusions are based on extensive clinical and experimental evidence and agree with my own findings.[2] All the laryngeal muscles are innervated unilaterally by the recurrent laryngeal nerve except the cricothyroid muscle, which is supplied by the corresponding external ramus of the superior laryngeal nerve. As the interarytenoid is a midline muscle, it is innervated by both the right and the left recurrent laryngeal nerve.

The findings of two other investigators, however, are often quoted as established facts. Exner[3] believed that each laryngeal muscle was bilaterally and doubly supplied by both the superior and the recurrent laryngeal nerves. More recent investigations discredit his results, however, because of the faulty experimental methods used in his work. The teaching of Luschka[4] is becoming more widely accepted (Judd, New and Mann,[5] Dilworth,[6] New,[7] Berlin and Lahey[8] and Nordland[9]), as it seems to explain best the clinical findings in laryngeal paralysis. This view differs from Ónodi's dictum only in that the interarytenoid

From the Department of Anatomy, University of Nebraska Medical College.

1. Ónodi, A.: Die Anatomie und Physiologie der Kehlkopfnerven, Berlin, Oscar Coblentz, 1902.

2. Lemere, F.: Innervation of the Larynx: I. Innervation of Laryngeal Muscles, Am. J. Anat. **51**:417 (Nov.) 1932.

3. Exner, S.: Die Innervation des Kehlkopfes, Sitzungsb. d. Akad. d. Wissensch. **89**:63, 1884.

4. Luschka, H.: Der Kehlkopf des Menschen, Tübingen, H. Laupp, 1871.

5. Judd, E. S.; New, G. B., and Mann, F. C.: Effect of Trauma on the Laryngeal Nerves, Ann. Surg. **67**:257 (March) 1918.

6. Dilworth, T. F. M.: The Nerves of the Human Larynx, J. Anat. **56**:48 (Oct.) 1921.

7. New, G. B.: Larynx in Diseases of Thyroid, Ann. Clin. Med. **1**:262 (Jan.) 1923.

8. Berlin, D., and Lahey, F. H.: Dissection of Recurrent and Superior Laryngeal Nerves, Surg., Gynec. & Obst. **49**:102 (July) 1929.

9. Nordland, M.: The Larynx as Related to Surgery of Thyroid, Surg., Gynec. & Obst. **51**:449 (Oct.) 1930.

muscle is considered as being innervated by both the recurrent and the superior laryngeal nerves. However, no one has ever produced contraction of any of the laryngeal muscles except the cricothyroid by stimulation of the superior laryngeal nerve. Therefore, I believe that the branches of the internal ramus of the superior laryngeal nerve which appear to innervate the interarytenoid muscle in reality pass on through to supply the underlying mucous membrane. There is no evidence that the sympathetic nervous system has anything to do with innervation of the laryngeal muscles, except perhaps indirectly through vasomotor disturbances.[2]

The first recorded observations on experimental paralysis of the laryngeal nerves were made by Galen some time between 160 and 200 A.D. He found that bilateral sectioning of the recurrent laryngeal nerve in animals caused aphonia and respiratory distress. Longet [10] and others discovered that young animals, especially cats, were susceptible to this paralytic dyspnea, which was occasionally severe enough to cause death. These interesting experimental observations have become more significant with the advent of modern surgical treatment of the thyroid gland. The aphonia and dyspnea which occasionally follow removal of the thyroid gland depend in some way on the close relationship of the laryngeal nerves to the thyroid gland, as has been emphasized by Berlin and Lahey,[8] Fowler and Hanson [11] and Nordland.[9] That these nerves might be cut, traumatized or compressed by contraction of the scar tissue subsequent to surgical treatment is apparent, but the effect on the glottis of paralysis of the various laryngeal nerves after operation—or any disease process—is not so obvious. It will be the purpose of this paper, therefore, to record the study of the changes in the glottic picture following experimental paralysis of the various laryngeal nerves as they might occur singly or in combination.

MATERIAL AND METHODS

The dog was chosen for the experiments because of the facility the animal lends to this particular type of investigation. Repeated observations were made as to the dependability of carrying results over from the dog to man. Binocular dissections of fifteen canine larynges were compared with binocular dissections of two human larynges. This comparison was supplemented by graphic reconstruction of a complete series of sections through a pup's larynx stained with hematoxylin and eosin.

The action of each laryngeal muscle in the dog was studied by isolating it on an otherwise stripped laryngeal skeleton and stimulating it electrically. This was

10. Longet, A.: Recherches sur les fonctions des muscles et nerfs du larynx, Gaz. méd. de Paris **9**:465 (July) 1841.

11. Fowler, C. H., and Hanson, W. A.: Surgical Anatomy of Thyroid Gland, Surg., Gynec. & Obst. **49**:59 (July) 1929.

necessary because of the lack of agreement which exists in current laryngologic teachings as to the part played by each muscle in modifying the glottic picture. The pattern of the laryngeal nerves and the innervation of the laryngeal muscles have already been described in the two previous papers of this series.

The effect on the glottic picture of sectioning the various laryngeal nerves in twenty adult dogs, seven of which were operated on and then observed for a month or more before being killed and examined, was next studied. By placing the dogs on an animal board in a supine position, an excellent view of the glottis could be obtained with a laryngeal speculum. The examination was usually made while the dog was coming out of light ether anesthesia. When a more prolonged observation was desired, the trachea was cut across just below the cricoid cartilage, and the cords were viewed from below. With this method a low intratracheal cannula can be inserted for continuous inhalation anesthesia. Light ether narcosis plus amytal was always used to eliminate the depressant effect of deep ether anesthesia on the abductor function of the glottis.[12]

The changes in the glottic picture during recovery from paralysis of the recurrent laryngeal nerve were studied by pinching the nerve with a hemostat. The dogs were examined with the laryngeal speculum at regular intervals until complete recovery had taken place. The effect that contracting scar tissue might have through pressure on the laryngeal nerves was studied by the accelerated process of subjecting the nerves to traumatizing agents such as drying or heat and cold. As the results agreed entirely with Semon's law, which is well established clinically, the less easily controlled method of reproducing contraction of scar tissue experimentally was not attempted.

ANATOMIC AND PHYSIOLOGIC BACKGROUND FOR INTERPRETATION OF PARALYSIS OF THE LARYNGEAL NERVES

The laryngeal structure in dogs (fig. 1, *A* to *E*) was found to correspond closely to that in man. The ventricular apparatus, however, is more highly developed in the dog, the labored respirations of which often demand an increased secretion of mucus for the protection of the vocal cords. The cuneiform cartilage is relatively very large, and the ventricular muscle associated with it is probably represented in man by the oblique part of the interarytenoid muscle. The ventricular sac also is relatively deep in the dog, which increases the normal tendency of the cords to approach the midline on deep inspiration.[13] These and other slight differences have only minor significance in the problem, and for all practical purposes the larynges of man and the dog are identical.

The actions of the individual laryngeal muscles were found by the experiments in direct stimulation to be as follows: The cricothyroid stretches the corresponding vocal cord, which tends to approach the midline or to become adducted. The thyro-arytenoid relaxes the vocal

12. Semon, F., and Horsley, V.: On an Apparently Peripheral and Differential Action of Ether on the Laryngeal Nerves, Brit. M. J. **2**:405, 1886.

13. Browne, Lennox: Throat and Nose and Their Diseases, ed. 5, London, Baillière, Tindall & Cox, 1899, p. 25.

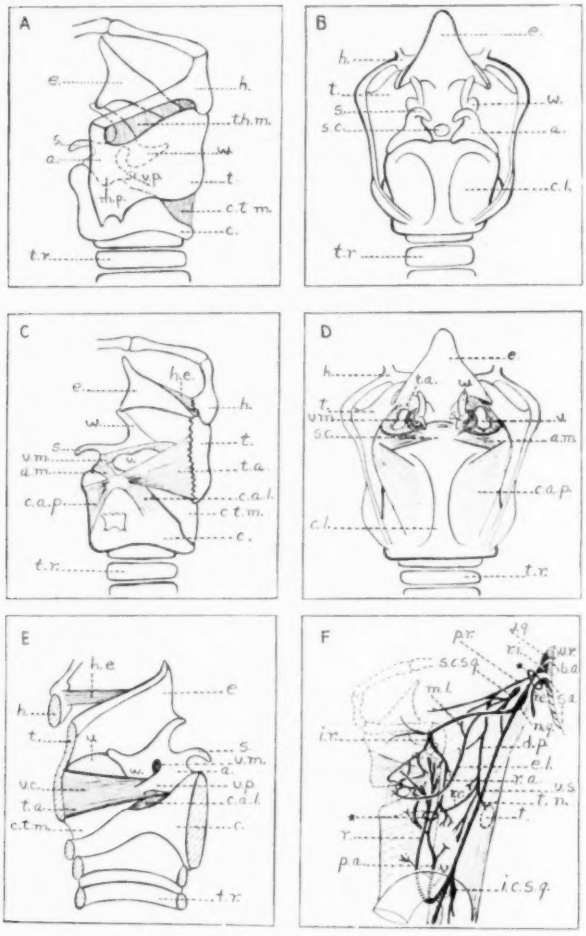

Fig. 1.—*A*, lateral view of the laryngeal skeleton in the dog. *B*, posterior view of the laryngeal skeleton in the dog. *C*, lateral view of the laryngeal muscles in the dog. *D*, posterior view of the laryngeal muscles in the dog. *E*, medial view of the laryngeal skeleton and muscles in the dog. In these parts, *a.* indicates arytenoid cartilage; *a.m.*, interarytenoid muscle; *c.*, cricoid cartilage; *c.a.l.*, lateral crico-arytenoid muscle; *c.a.p.*, posterior crico-arytenoid muscle; *c.l.*, lamina of cricoid cartilage; *c.t.m.*, cricothyroid membrane; *e.*, epiglottis; *h.*, hyoid cartilage; *h.e.*, hyo-epiglottic muscle; *m.p.*, muscular process of arytenoid cartilage; *s.*, cartilage of Santorini (corniculate); *s.c.*, sesamoid cartilage; *t.*, thyroid cartilage; *t.a.*, thyro-arytenoid muscle; *t.h.m.*, thyrohyoid membrane; *t.r.*, tracheal ring; *v.*, ventricle; *v.c.*, vocal cord; *v.m.*, ventricular muscle; *v.p.*, vocal process of arytenoid cartilage; *w.*, cartilage of Wrisberg (cuneiform). *F*, pattern of the laryngeal nerves in the dog. In this part, *b.a.* indicates bulbar roots of accessory nerve; *d.p.*, descending pharyngeal nerve; *e.l.*, external branch of superior laryngeal nerve; *i.c.s.g.*, inferior cervical sympathetic ganglion; *i.r.*, internal ramus of superior laryngeal nerve; *j.g.*, jugular ganglion; *m.l.*, medial laryngeal nerve; *n.g.*, nodose ganglion; *p.a.*, pararecurrent nerve; *p.r.*, pharyngeal ramus of vagus; *r.*, recurrent nerve; *r.a.*, ramus anastomoticus; *r.c.*, ramus communicans of descending pharyngeal nerve; *r.e.*, external ramus of eleventh nerve; *r.i.*, internal ramus of eleventh nerve; *s.a.*, spinal accessory roots; *s.c.s.g.*, superior cervical sympathetic ganglion; *t.*, thyroid gland; *t.n.*, thyroid nerve; *v.r.*, vagus roots; *v.s.*, vagosympathetic trunk. *, lying in same sheath, but separable or not fused.

cord and also adducts it. The lateral crico-arytenoid adducts the anterior or membranous half of the vocal cord. The posterior crico-arytenoid abducts or swings the vocal cord out laterally, dilating the glottis on the same side. The interarytenoid closes the cartilaginous or posterior half of the glottis. The effect of the extrinsic laryngeal muscles on the glottic picture is negligible.

The pattern of the laryngeal nerves in the dog (fig. 1 *F*) differs briefly in two particulars from that in man. First, the recurrent laryngeal nerve is divided into two parts, the recurrent proper, which innervates the laryngeal muscles alone, and the pararecurrent, which innervates the trachea and the esophagus. The second difference is that in half the dogs examined the innervation of the cricothyroid muscle was derived from a branch of the pharyngeal ramus of the vagus, called the median laryngeal nerve, rather than from the external ramus of the superior laryngeal nerve. Recognition of these differences is essential if one is to avoid gross errors in the interpretation of experimental findings. For the sake of simplicity, however, and also because the median laryngeal nerve usually joins the external ramus of the superior laryngeal nerve before entering the cricothyroid muscle, the innervation of the cricothyroid muscle will be referred to as coming from the external ramus of the superior laryngeal nerve.

The normal physiology of the larynx must be understood before attempting to explain the pathologic reactions created by paralysis of the different laryngeal muscles. According to Negus,[14] there are three main functions of the larynx: protection in swallowing, the maintenance of a patent air way and vocalization. The first function depends on the integrity of all the intrinsic laryngeal muscles except the cricothyroid and the posterior crico-arytenoid. Respiration depends only on the latter muscle, while vocalization requires all but this muscle. The normal glottic picture varies from full adduction (fig. 2 *B*) with the cords in apposition, as in swallowing and vocalization, to full abduction (fig. 2 *A*), as in deep inspiration. In quiet breathing, complete denervation or death, the cords assume the cadaveric or resting position, i. e., half way between the two extremes given (fig. 2 *C*).

EFFECT OF SECTIONING THE VARIOUS LARYNGEAL NERVES ON THE GLOTTIC PICTURE

Sectioning of both recurrent laryngeal nerves causes paralysis of all the laryngeal muscles except the cricothyroids, which tend to stretch the cords anteroposteriorly and draw them toward the midline. This produces a partially adducted position of the glottis (fig. 2 *D*), which

14. Negus, V. E.: The Mechanism of the Larynx, London, William Heinemann, Ltd., 1929.

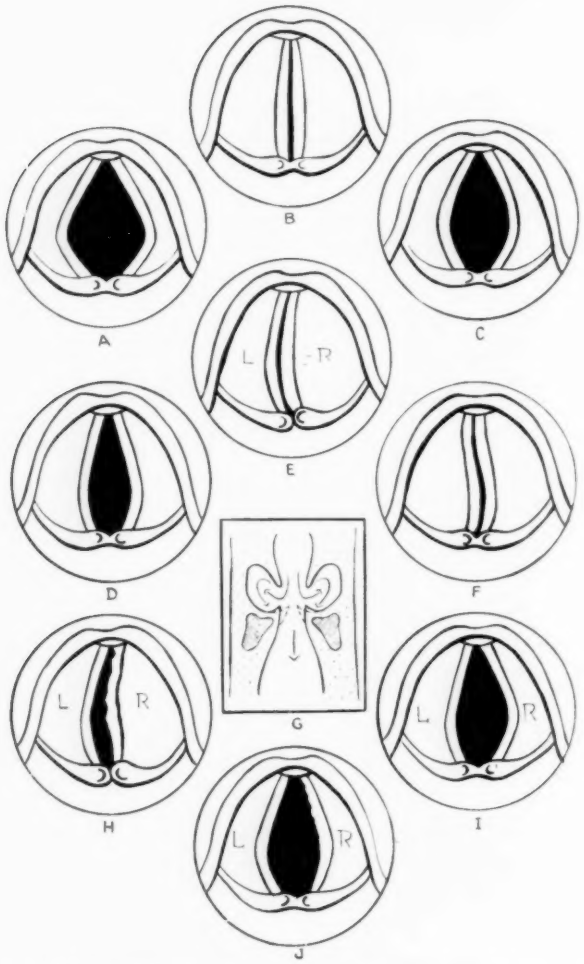

Fig. 2.—*A*, full abduction of the vocal cords on deep inspiration. *B*, full adduction of the vocal cords as in phonation. *C*, cadaveric position of the vocal cords as in complete paralysis of the cords, quiet respiration or death. *D*, partially adducted position of the vocal cords accompanying bilateral paralysis of the recurrent laryngeal nerves. *E*, unilateral paralysis of the recurrent laryngeal nerve on the left, with the right cord crossing the midline on attempted phonation. *F*, bilateral paralysis of the external rami of the superior laryngeal nerves with relaxation of the vocal cords on attempted phonation due to paralysis of the cricothyroids. *G*, diagram of cross-section through the larynx, showing approximation of the vocal cords during inspiration. This increases the dyspnea which accompanies complete or partial paralysis of the vocal cords. *H*, complete paralysis of the recurrent laryngeal nerve on the left, with the cord in the partially adducted position. Paralysis of the external ramus of the superior laryngeal nerve on the right, with relaxation of the right cord which crosses the midline on attempted phonation. *I*, bilateral paralysis of the recurrent laryngeal nerves plus paralysis of the external ramus of the superior laryngeal nerve on the right. The left cord is in the partially adducted position, and the right is in the cadaveric position. *J*, bilateral paralysis of the external rami of the superior laryngeal nerves plus paralysis of the recurrent laryngeal nerve on the left. Both cords are in the cadaveric position, with relaxation of the cord on the right.

is midway between full adduction and the cadaveric position that has always previously been associated with this type of paralysis. The partial adduction, together with the absence of normal inspiratory abduction resulting from paralysis of the posterior crico-arytenoid muscles, causes the most marked dyspnea found in paralyses of the laryngeal nerves. The dyspnea may result in immediate asphyxia, or it may be marked only on exertion, depending on the relative size of the glottis and the oxygen requirements of the individual dog. Young animals especially are susceptible to this paralytic dyspnea, because of the relatively large oxygen requirement and the small size of the glottic opening. The least protection in swallowing is found in this type of paralysis, because all the laryngeal muscles are inactive except the cricothyroids, which increase the anteroposterior diameter of the glottis rather than narrow it as is usual in swallowing. Although the extrinsic laryngeal muscles have no effect on the glottic picture, they aid materially in the mechanism of swallowing by drawing the larynx upward and forward under the epiglottis. The cricothyroid muscles, by stretching and adducting the cords, make coughing possible. Coughing helps to free the air passage of foreign material and is a prominent feature of this type of paralysis. Vocalization is coarse or impossible, as the cricothyroid muscles play only a small part in the mechanism of the production of voice.

Sectioning of one recurrent laryngeal nerve causes some dyspnea with exertion, owing to the partial adduction of the paralyzed cord and its inability to abduct on inspiration. There is only a little hoarseness on phonation, for the healthy cord crosses the midline to appose itself to the paralyzed cord (fig. 2*E*). Swallowing is not interfered with, mainly because of the bilateral innervation of the interarytenoid muscle, which closes the posterior portion of the glottis.

Bilateral sectioning of the external rami of the superior laryngeal nerves has little or no effect on the glottic picture. The tension of the vocal cords is lost, however, with paralysis of the cricothyroid muscles, and the cords wrinkle on attempted phonation (fig. 2*F*). This results in coarse phonation. There is no difficulty in respiration or in swallowing, as the paralyzed cricothyroid muscles play no part in these functions of the larynx. One-sided section causes slight hoarseness on vocalization, owing to partial relaxation of the corresponding vocal cord. As in bilateral section, there is no dyspnea or difficulty in swallowing.

Bilateral sectioning of the external rami of the superior laryngeal nerves and the recurrent laryngeal nerves results in complete paralysis of all the intrinsic laryngeal muscles. The glottis assumes the cadaveric position (fig. 2*C*), and vocalization is impossible. The difficulty in swallowing is not quite so great as in straight bilateral paralysis of the

recurrent laryngeal nerves, because there is no unopposed cricothyroid muscle to stretch the glottis anteroposteriorly. The extrinsic laryngeal muscles help to draw the larynx forward under the arytenoid cartilage during the act of swallowing. True coughing is lost, as the cords cannot be apposed. With the glottis in the cadaveric position, breathing is not difficult as long as the dog is at rest. The slightest exertion, however, causes dyspnea, because the normal inspiratory abduction of the cords is lost. The mechanical effect of convex air currents is no small factor in the production of the dyspnea occurring with the flaccid paralysis of complete denervation. During deep inspiration, the incoming air tends to appose the cords, which act as valves owing to the ventricular sac behind them (fig. 2 *G*).

Sectioning of the external ramus of the superior laryngeal nerve on one side and of the recurrent laryngeal nerve on the other side causes paralysis of the cricothyroid muscle on the one side, with relaxation of the corresponding cord, and a partially adducted position of the vocal cord on the other side, with paralysis of abduction. This makes vocalization difficult or impossible, as one cord is relaxed and the other cannot be completely adducted (fig. 2 *H*). There is very little respiratory distress, as full abduction is present on one side. Swallowing is little disturbed, as the interarytenoid muscle closes the glottis behind.

Sectioning of both recurrent laryngeal nerves and of the external ramus of the superior laryngeal nerve on one side causes complete paralysis of the cord on the latter side, the other cord being in a partially adducted position (fig. 2 *I*). The functional result is predominantly that of bilateral section of the recurrent laryngeal nerves, except that the dyspnea is not quite so severe, owing to the relaxation of the cricothyroid muscle on the one side. Vocalization is practically impossible.

Sectioning of the external rami of both superior laryngeal nerves and of the recurrent laryngeal nerve on one side results in complete paralysis on the latter side and relaxation of the cord on the other side (fig. 2 *J*). There is little respiratory distress, as the relaxed cord may be abducted. Difficulty in swallowing is not marked, because the interarytenoid muscle closes the larynx posteriorly. Vocalization is very poor, owing to the relaxation of the cords on both sides and the inability to adduct the cord on the one side.

Sectioning of the internal ramus of the superior laryngeal nerve has no effect on the glottic picture. The sensory disturbances caused by such a section are well known.

After pinching the recurrent laryngeal nerve with a hemostat, there is complete paralysis of all the muscles supplied by this nerve. The corresponding vocal cord assumes the partially adducted position already described. Complete recovery takes place in from one to three

months. The adductor function is restored before the abductor, or respiratory, function of the glottis. Dyspnea is most marked at first, because of the partially adducted position of the cord, but as the muscles antagonistic to the cricothyroid regain their tone, this gradually passes off until finally the posterior crico-arytenoid muscle recovers, and normal inspiratory abduction is regained. This is the reverse of Semon's law, which obtains in partial and slowly produced paralysis of the recurrent laryngeal nerve, in which the abduction, or respiratory, function is the first to drop out before complete paralysis sets in. Incomplete trauma to the recurrent laryngeal nerves invariably resulted in this preliminary loss of abduction alone.

COMMENT

The immediate glottic picture produced by complete sectioning of the various laryngeal nerves need no further explanation. Incomplete paralyses and latent changes in the position of the cords, however, require more detailed consideration. Semon [15] and Russell [16] showed that incomplete paralysis of the recurrent laryngeal nerve causes a loss of abduction only. It has just been observed that there is a stage between complete paralysis and complete recovery in which the same condition exists.

Therefore, in beginning paralysis, as from contracting scar tissue, or in beginning recovery, as after pinching the recurrent laryngeal nerve, there may result a loss of abduction alone, with the glottis in the cadaveric position. This explains the observations of New [7] that in some cases the primary cadaveric position passes over to an adducted position after from three to six months (incomplete paralysis of the recurrent laryngeal nerves becoming complete). Isolated abductor paralysis causes dyspnea only on exertion and has no effect on swallowing or phonation.

The muscles of the larynx are always in a delicate state of balance, and when one group is paralyzed the antagonistic group overacts. This is especially true in paralysis of the recurrent laryngeal nerves in which the unparalyzed cricothyroid muscles stretch and adduct the vocal cords. After some time, however, the cricothyroid muscles undergo atrophy of disease, because they no longer have to pull against the antagonistic thyro-arytenoid muscles, which have been paralyzed. This phenomenon was studied experimentally by Grossmann,[17] and explains why in some

15. Semon, F.: Proclivity to Disease of Abductor Fibers, Arch. Laryng. **2**:197 (July) 1881.

16. Russell, J. S. R.: The Abductor and Adductor Fibers of the Recurrent Laryngeal Nerve, Proc. Roy. Soc., London **51**:102 (March 31) 1892.

17. Grossmann, M.: Funktionelle Beziehung der Kehlkopfmuskeln, Arch. f. Laryng. u. Rhin. **18**:463, 1906.

cases the dyspnea accompanying paralysis of the recurrent laryngeal nerves is lessened with the passing of time. The cricothyroid muscles atrophy, and a cadaveric position of the cords is substituted for a partially adducted position.

It is difficult to understand why the internal ramus of the superior laryngeal nerve has been considered as playing a part in these paralyses. It is true that some of its fibers run through the interarytenoid muscle. However, stimulation experiments irrefutably demonstrate that these fibers merely traverse the muscle on their way to the underlying mucosa. It is significant to find that Exner, whose experiments on degeneration are used to support this theory of innervation, had finally to admit to Grossmann that his results were in error and that the internal ramus of the superior laryngeal nerve carried no motor fibers. The edema and swelling of the cords incident to the trauma in this region, as well as the tracheal collapse, must not be overlooked as frequent causes of respiratory distress immediately after thyroidectomy.

The physiologic effect of sectioning the various nerves is not always typical. Human beings have been known to vocalize with the aryepiglottic folds after removal of the vocal cords. Even in the dogs used in these experiments the amount of dyspnea and the ability to vocalize after identical lesions has been produced varied greatly.

SUMMARY

Comparison of the structure, function and innervation of the larynx in man and in the dog indicates that experimental observations on the latter may reasonably be applied to the interpretation of clinical findings in man. The presence of a pararecurrent and median laryngeal nerve in dogs, however, must be recognized if errors in the interpretation of experimental results are to be avoided. The ventricular sac in the dog also differs from that in man, in that it is comparatively larger. This produces a valvelike action of the vocal cords with a greater tendency toward adduction on inspiration than is usual in man.

The exact action of each laryngeal muscle in the dog was studied by isolating it on an otherwise stripped laryngeal skeleton and stimulating it electrically. In general, all the intrinsic laryngeal muscles constrict the glottis except the posterior crico-arytenoid, which dilates the glottis. The effect of the extrinsic laryngeal muscles on the glottis is negligible except as an aid in swallowing.

In two previous papers the intrinsic muscles of the larynx have been described as being innervated by the recurrent laryngeal nerve, except the cricothyroid, which is innervated by the external ramus of the superior laryngeal nerve. This innervation is unilateral except

for the interarytenoid muscle, which is supplied by both recurrent laryngeal nerves. The internal ramus of the superior laryngeal nerve contains no motor fibers, and its section in no way alters the glottic picture.

With this anatomic and physiologic background established, the effect on the glottis of sectioning the various laryngeal nerves was next studied in dogs. The glottic picture and functional disability of the larynx observed in each type of paralysis of the laryngeal nerve were correlated as much as possible with the action of the muscles involved. The adductor and abductor functions of the larynx were also studied in relation to the differential effects of beginning paralysis and beginning recovery from complete paralysis of the recurrent laryngeal nerve.

CONCLUSIONS

1. Sectioning of the recurrent laryngeal nerve produces a partially adducted position of the corresponding vocal cord, which is stretched anteroposteriorly by the unparalyzed cricothyroid muscle. This partially adducted position of the cord is decidedly different from the cadaveric position which has always been associated with this type of paralysis. Abduction of the paralyzed cord is impossible. Bilateral sectioning causes severe dyspnea and difficulty in swallowing and phonation.

2. Sectioning of the external ramus of the superior laryngeal nerve produces relaxation of the corresponding vocal cord owing to paralysis of the cricothyroid muscle on that side. This interferes with phonation, but there is no dyspnea or difficulty in swallowing.

3. Sectioning of both the recurrent nerve and the external ramus of the superior laryngeal nerve on one side results in complete paralysis of the corresponding vocal cord, which assumes the cadaveric position. With bilateral sectioning of both nerves, there result absolute aphonia and dyspnea on exertion. Swallowing is made possible by the action of the extrinsic laryngeal muscles.

4. Sectioning of the recurrent nerve on one side and of the external ramus of the superior laryngeal nerve on the other side produces a partially adducted position of the cord on the one side and relaxation of the opposite cord. There is hoarseness of phonation, but very little dyspnea or difficulty in swallowing.

5. Sectioning of both recurrrent laryngeal nerves and of the external ramus of the superior laryngeal nerve on one side produces complete paralysis of the cord on the latter side, with a partially adducted position of the cord on the other side. Phonation is impossible, and there are marked dyspnea and difficulty in swallowing.

6. Sectioning of the external rami of both superior laryngeal nerves and of the recurrent laryngeal nerve on one side produces complete paralysis of the cord on the latter side with relaxation of the opposite cord. Vocalization is coarse or impossible. There are some dyspnea and difficulty in swallowing.

7. Partial paralysis of the recurrent laryngeal nerve, including beginning recovery from complete paralysis, leads to isolated abductor paralysis with dyspnea on exertion.

8. The severe dyspnea of complete bilateral paralysis of the recurrent laryngeal nerves may diminish with time, owing to atrophy of disuse in the unaffected cricothyroid muscles. This produces a glottic picture and a functional result identical with those of complete denervation of the laryngeal muscles.

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CHRONIC PARANASAL SINUS INFECTION

RELATION TO DISEASES OF THE LOWER RESPIRATORY TRACT

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Acute inflammation of the paranasal sinuses is probably as common as the common cold. In most instances it is probably as transient. Chronic sinusitis is, however, also an extremely common disease. While at times a purely local condition, it is often associated with other diseases of the respiratory tract, diseases to which the sinus condition may be secondary, or in the causation of which the sinus disease may play a part.

A prime and obvious factor in the production of chronic sinus disease is bacterial invasion. A second well recognized factor is the presence of an intranasal anatomic abnormality, such as marked septal deflection, which interferes with the free drainage of the sinuses. Repeated infection associated with the anatomic defect serves in time to destroy the ciliated epithelium of the sinuses, putting out of action an important mechanism in normal drainage of the sinuses, and thereby tending to make the disease chronic. A further and most important factor in the production of chronic sinusitis is the swelling of the nasal mucosa which is so characteristic of nasal allergy.

We shall present some clinical data which appear to have a bearing on the relation of chronic infection of the paranasal sinuses to diseases of the lower respiratory tract.

Figures for the incidence of sinus disease occurring in other diseases of the respiratory tract have no absolute value unless they are compared with similar figures obtained in a group of so-called normal controls. Furthermore, each set of figures must be obtained in groups of persons from the same geographic region, for the incidence of sinus disease varies widely with locality. Several years ago we [1] studied such a control

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1. Kern, R. A., and Schenck, H. P.: The Relative Efficiency of the Clinical and the Roentgenologic Methods for Sinus Disease Diagnosis, Am. J. M. Sc. **178:** 168, 1929.

group of fifty persons (table 1). They gave no history of respiratory disease occurring within six months, of susceptibility to colds or of frank sinus disease, and their ages fell proportionately into the same decades as those of our first two hundred asthmatic patients. A group so selected may well be considered as better than average. Yet 72 per cent gave roentgen evidence of abnormality of the sinuses, and in 26 per cent the abnormality was clinically detectable. There is every reason to believe that these abnormal findings may be evidence of sinus disease in the past rather than of active sinusitis. But it seems to be clear that three fourths of the population have or have had at some time in their lives a protracted or a severe sinus infection.

TABLE 1.—*Incidence of Sinus Disease in So-Called Normal Controls*

Persons examined	50
Persons found with abnormal sinuses on clinical examination	13 (26%)
Abnormal sinuses per person (average)	2.34
Persons found with abnormal sinuses on roentgen examination	36 (72%)
Abnormal sinuses per person (average)	3.98

TABLE 2.—*Incidence of Sinus Disease in Bronchiectasis*

	Bronchiectasis	Controls
Cases	50	50
Positive clinical findings		
Patients	40 (80%)	13 (26%)
Involved sinuses per patient (average)	4.32	2.34
Positive roentgen findings		
Patients	50 (100%)	36 (72%)
Involved sinuses per patient (average)	5.32	3.98

Chronic diseases of the lower respiratory tract that are attended by purulent sputum in large amounts show an overwhelming incidence of chronic sinus infection. In our experience, both the incidence and the degree of sinus involvement are higher in bronchiectasis than in any other condition (table 2). All of our fifty patients had roentgen indications of, and 80 per cent showed clinical evidence of, active sinus disease. In most instances there was a pansinusitis, and the average number of sinuses involved per patient was very high. Sinus disease is almost as common in chronic pulmonary abscess and in chronic ulcerative tuberculosis with cavitation, mixed infection and profuse purulent sputum.

Whether the sinus disease is primary or secondary in bronchiectasis cannot usually be determined in the individual instance. Undoubtedly, chronic purulent sinusitis may be the starting point for chronic bronchial infection and subsequent bronchiectasis. But it is also certain that the constant spraying of the nasal chambers by coughed-up purulent sputum serves not only to maintain an existing sinus infection but to infect

sinuses not already involved. We have been able to observe some patients with posttonsillectomy abscess of the lung in whom the sinuses were originally clear on both clinical and roentgen examination, but in whom within a short time (weeks) a purulent pansinusitis developed.

From the practical standpoint it is obvious that the treatment of such chronic suppurative disease of the lower respiratory tract is doomed to failure if it does not include the thorough treatment of the sinus infection.

Significant points in this group of cases are: the constancy of marked clouding of the sinuses on roentgen examination and on transillumination, and the usual finding of a red, congested nasal mucosa.

While we are concerned primarily with chronic sinus disease, it is well to bear in mind a rather frequent subacute bronchial infection that seems to be dependent on an acute sinusitis. It has been described under the name of bronchosinusitis.[2] In these patients, usually young adults

TABLE 3.—*Incidence of Sinus Disease in Pollinosis*

	Pollinosis	Controls	Bronchiectasis
Cases	50	50	50
Positive clinical findings			
Patients	29 (58%)	13 (26%)	40 (80%)
Involved sinuses per patient (average)	3.0	2.34	4.32
Positive roentgen findings			
Patients	37 (74%)	36 (72%)	50 (100%)
Involved sinuses per patient (average)	2.3	3.98	5.32

or adolescents, after an acute cold in the head a cough develops with fever and malaise for a few days. These symptoms ameliorate, but do not clear up entirely. Cough and purulent sputum persist, and there may be a mild leukocytosis and an afternoon rise of temperature. Physical signs are usually confined to a few râles in one lower lobe, with minimal or no changes on percussion. Roentgen examination shows greatly increased trunk shadows near the hilus of the affected lobe and perhaps even some central peribronchial consolidation. An associated acute purulent sinusitis may be silent and is therefore often overlooked. The cure of the sinusitis is followed by prompt clearing of the pulmonary lesion.

If patients with seasonal hay fever or pollen asthma are examined during the pollen season, plenty of evidence is found that the sinus mucosa is involved as well as the nasal mucosa. Proetz [3] clearly showed that on exposure to an allergen the sinus mucosa may swell to the point

2. Wasson, W. W.: Bronchosinusitis Disease, J. A. M. A. **93**:2018 (Dec. 28) 1929.

3. Proetz, A. W.: Displacement Method in Sinus Diagnosis and Treatment: Its Advantages and Limitations, Tr. Am. Laryng. A. **52**:121, 1930.

of practically obliterating the sinus cavity. Yet if these patients who are subject to pollinosis are examined just before or soon after the pollen season,[4] we find roentgen incidence of sinus abnormality that is no greater than, if as great as (fewer abnormal sinuses per patient), that in our controls (table 3). The significant point here, we believe, is the fact that the pollinosis occurs at the time of year when infections of the upper respiratory tract are at a minimum. As soon as the short pollen season is over, the mucosal swelling subsides because there is no complicating infection. (Obviously, too, infection of the sinuses plays no major part in hay fever and pollen asthma.)

Patients with perennial nasal and bronchial hypersensitiveness show an extremely high incidence of abnormality of the sinuses, both clinically and in roentgenograms. Here, obviously, the combination of infection and blockage of the sinus ostia by a swollen mucosa has brought about

TABLE 4.—*Incidence of Sinus Disease in Bronchial Asthma and Vasomotor Rhinitis*

	Asthma	Vasomotor Rhinitis	Controls	Pollinosis	Bronchiectasis
Cases	400	40	50	50	50
Positive clinical findings					
Patients	279 (70%)	31 (77%)	13 (26%)	29 (58%)	40 (80%)
Involved sinuses per patient (average)	3.17	4.3	2.34	3.0	4.32
Positive roentgen findings					
Patients	350 (87.5%)	36 (90%)	36 (72%)	37 (74%)	50 (100%)
Involved sinuses per patient (average)	4.18	3.42	3.98	2.3	5.32

chronic sinus disease. In many instances there develops a third factor, namely, sensitization to bacterial protein. The question as to the time relation or the sequence of these factors—infection, mucosa swelling, bacterial allergy—is a vexing one and not to be answered categorically. It is our feeling, however, based on clinical experience, that in a minority of cases there is first an infection, then an infectious swelling of the mucosa occurs, and eventually, if the patient is of allergic heredity, there can supervene a true bacterial hypersensitiveness. The last mentioned condition continues to keep the mucosa in a swollen state, but the swelling is no longer inflammatory (red, congested) but allergic (pale, edematous, *hyperplastic*). In the majority of cases, however, we believe that protracted exposure to inhaled allergens produces a chronically swollen mucosa (an allergic swelling). This state, continued into the seasons of the year when respiratory infections are common, prepares the way for bacterial invasion of the sinuses. This in turn may be

4. Kern, R. A., and Donnelly, J. C.: The Incidence and Rôle of Paranasal Sinus Disease in Hay Fever, J. Allergy **3**:172, 1932.

followed by true bacterial hypersensitivity. In the majority, then, the sinus disease develops secondarily to the allergy (asthma, vasomotor rhinitis).

But whether the sinus infection is primary or secondary in point of time relation to the asthma, the sinus disease is a potent factor for further trouble, and for these reasons: It renders the nasal mucosa more vulnerable to old allergens; it may pave the way for sensitization to new inhalants or to bacteria, and it perpetuates the bronchial infection.

From the practical standpoint, it is important to remember that the avoidance of those extraneous substances to which the patient is sensitive is often followed by a considerable or complete subsidence of mucosal swelling. This in turn may lead to spontaneous drainage and to cure of the sinus infection. Only when this measure has failed is operation on the sinuses indicated.

The mucosal swelling which is almost constantly present in allergic patients accounts for the frequent finding of hazy rather than opaque sinuses on transillumination, and of hazy rather than cloudy sinuses in the roentgen film.

We shall mention briefly a further important and, we believe, original observation. It has to do with the etiology of so-called mucous nasal polyps and is discussed by us at length in a paper that will soon be published.[5] Polyps are extremely common in allergic states. In a series of five hundred asthmatic patients we found polyps in nearly 30 per cent. On the other hand, in examining the nasal chambers of many patients with nonallergic diseases (e. g., bronchiectasis), we rarely found nasal polyps, even in the presence of the most extensive sinus infection. We extended our observations by examining several hundred patients with nonallergic diseases. Polyps were rarely encountered, but in each of the few instances in which they were found, they proved to be in a hypersensitive person with obvious personal and familial allergy. We have come to the conclusion that mucous nasal polyps occur only in the presence of an underlying hypersensitiveness. We believe, therefore, that the finding of nasal polyps should always suggest the further study of the patient from the standpoint of allergy.

5. Kern, R. A., and Schenck, H. P.: Allergy a Constant Factor in the Etiology of So-Called Mucous Nasal Polyps, J. Allergy **4**:485 (Sept.) 1933.

SEVERE DEAFNESS IN ADULTS

A CLINICAL STUDY

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During the past year we have made a clinical study of severe deafness in adults in an attempt to add a few definite facts to our meager store of knowledge concerning progressive deafness. How important is chronic sinus infection or nasal obstruction in the etiology of progressive deafness? How many adults with a severe defect in hearing suffer from primary nerve deafness and how large a proportion from obstruction to sound conduction? Is a combination of factors responsible for severe deafness in a considerable number of cases? How large a proportion of severely deafened adults suffer from otosclerosis? Finally, how many severely deafened adults have lost their hearing as the result of middle ear disease which began in childhood and which might have been prevented? These and other causes for severe deafness have long been recognized, but accurate statistics on the relative importance and frequency of each of these factors have not been available. Such statistics are necessary before any intelligent program for the prevention of deafness can be formulated.

Several years ago an investigation of profound deafness in children was carried out under the auspices of the National Research Council. More than 5,000 children in the public institutions for the deaf were examined. The results of this study are reviewed as a contrast to our findings for adult deafness.

Sixty per cent of the children are congenitally deaf, while 40 per cent lost their hearing during childhood. This 40 per cent with acquired deafness is of particular interest. Nearly all lost their hearing abruptly during a severe attack of cerebrospinal meningitis, measles, scarlet fever, pneumonia, influenza or whooping cough (fig. 1). Deafness from meningitis is frequently due to an invasion of the labyrinth

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through the internal auditory meatus, but deafness from the other acute infectious diseases is nearly always the result of a toxic neuritis of the eighth nerve. Approximately a third of these children had an associated otitis media, yet in only a few was it possible to ascribe the deafness to the otitis media. The majority of profound acquired deafness in childhood is due to a toxic neuritis of the nerve of hearing, occurring during an acute infectious disease. It was also found that most of these children lost their hearing during the first three years of life. Apparently, with advancing age, the eighth nerve becomes less susceptible to a toxic neuritis. The conclusion was that the prevention of profound acquired deafness in childhood lies in the prevention and prompt treatment of the acute infectious fevers, particularly during the first few years of life.

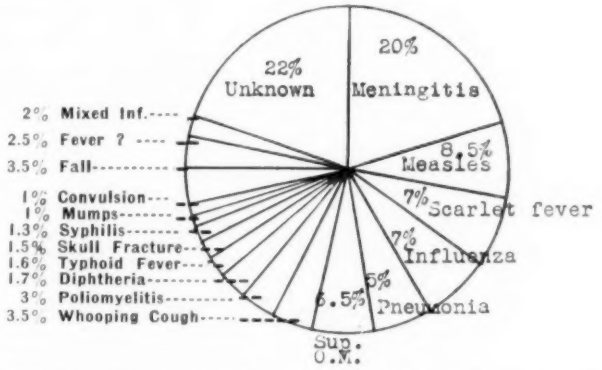

Fig. 1.—Etiology of acquired deafness in 5,000 children from the public institutions for the deaf.

We have limited the present study to adults whose deafness is severe enough to constitute a handicap. These are the people to whom deafness is a serious social and economic problem. It is for these people that the Leagues for the Hard of Hearing have been organized. Children afflicted with profound deafness are segregated in public institutions for the deaf, but severely deafened adults are not segregated, so that our first problem was to collect a group which would accurately represent severe adult deafness. It seemed to us that the membership of the Leagues for the Hard of Hearing represent as fair a cross-section of severe adult deafness as can be found.

We first studied a group of 60 volunteers from the Chicago League for the Hard of Hearing. Next we added 40 consecutive cases from our private practice. Finally, an opportunity arose to examine the members of the League for the Hard of Hearing in Washington, D. C. Owing to the able and enthusiastic cooperation of the president of

this organization, Mrs. Katherine Madden, we were able to include more than two thirds of all the members of the Washington League.

A complete history was obtained from each person following the outline shown in figure 2. A family history of deafness was obtained

NAME: AGE: SEX: MARITAL STATE:

ADDRESS: PHONE: MEMBER OF LEAGUE?

HISTORY OF DEAFNESS IN FAMILY:

	Members Deaf	Degree	Character of Onset	Time Deafness Began
Paternal history				
Maternal history				

HISTORY OF DEAFNESS IN PATIENT:

Age at time of onset: Mode of onset: Rapid / Insidious

Unilateral Bilateral

Previous ear trouble—
In childhood:
In adult life:

Nature of trouble: Discharge from ears
Recurring earache
Deafness

Did previous ear trouble persist until onset of present difficulty or was there a period when ear trouble complained of in childhood definitely subsided before onset of present difficulty?

Tinnitus: Constant or intermittent:
Duration:
Character:

Paracusis:

Use of the phone:

Has patient availed himself of use of electrical hearing device?
How much does hearing seem to be depressed during acute head colds?
What is and has been state of general health?
What illnesses have occurred, and their effect on hearing (including history of Paget's disease in self or in family)?
Pregnancy and its effect on deafness?
Vertigo?
What treatments or operations has patient had for deafness, and results?

Fig. 2.—History outline used in the examination.

in detail. Especial emphasis was placed on the relation of the present deafness to otic disease in childhood. Did the patient have earaches, a discharge from the ears or a defect in hearing as a child? If so, did this trouble progress to the present deafness or did the hearing

return to normal for a period before the onset of the present progressive deafness?

An examination of the nose and throat was then made (fig. 3), with a detailed description of the tympanic membranes. Finally, the hearing was tested for the whispered voice and with tuning forks. Audiograms were made in about 60 per cent of all the cases examined, the 1*A* Western Electric audiometer being used. However, the audiogram was found to be of no particular value for diagnosis in differentiating the cases of nerve deafness from those of conduction deafness. For example, figure 4 suggests nerve deafness. This patient had typical

EXAMINATION OF PATIENT:

Nasal passages:
Transillumination:
Nasopharynx:
Tonsils:
Drum membranes: Right Left
Patency of tubes: 1. By politzerization
2. By catheter
3. Effect on hearing

Functional tests:

Whispered voice	R		L
Weber *A*	c	Schwabach	A
C_1R	L	C R	L
A R	L	c^4R	L
Rinne R	L	GW R	L

Turning tests: Duration and character of nystagmus
Pointing tests

OCCUPATION OF PATIENT:

CONCLUSIONS:

Fig. 3.—Outline of the examination.

otosclerosis with primary fixation of the stapes. Figure 5 suggests conduction deafness, yet the tests with tuning forks showed a primary defect of the nerve due to senile changes.

As a result of these examinations, it was not difficult to arrive at a diagnosis in most of the cases. In a few of those with primary nerve deafness the etiology remained in doubt.

The last group examined was, perhaps, the most representative of severe adult deafness, since it included the majority of the membership of the Washington League for the Hard of Hearing. This league has approximately 100 members. Seventy of these were examined, but 5 proved to have only a slight defect, leaving 65 adults with severe deafness suitable for this investigation. For convenience, I have grouped the cases according to whether the tests for hearing showed

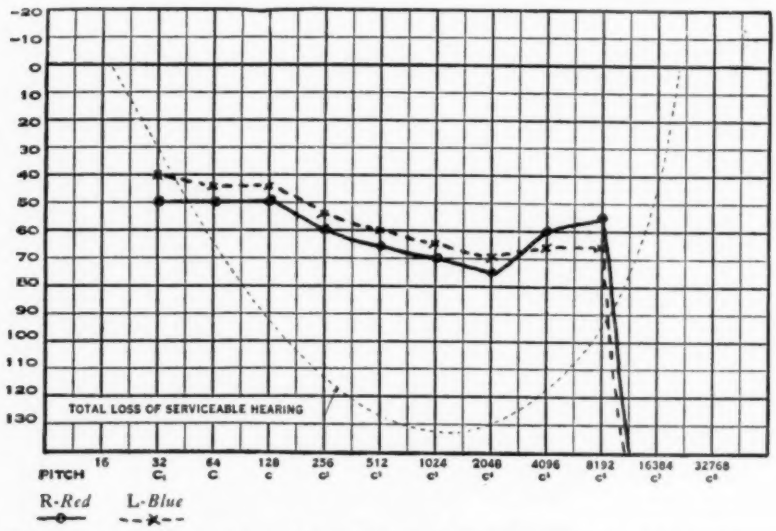

Fig. 4.—Audiogram of a patient (Mrs. J. P. Koch), aged 53, with otosclerosis. The sound of 32 and 64 double vibrations was not heard; hearing of 2,048 double vibrations was much shortened. The hearing for the Schwabach test was prolonged. The Rinne reaction was negative.

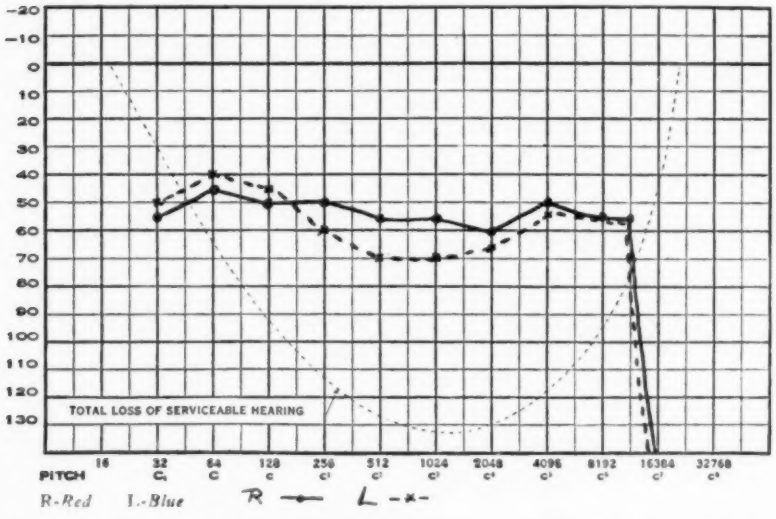

Fig. 5.—Audiogram of a patient (Mrs. A. J. Baer), aged 68, with primary nerve deafness. The length of time 32 double vibrations were heard was slightly shortened; 2,048 double vibrations, moderately shortened. The hearing for the Schwabach test was shortened. The Rinne reaction was positive.

a primary nerve deafness or obstruction to sound conduction. The essential findings in each case have been condensed and tabulated.

SUMMARY OF CASES

There were 11 patients showing the tuning fork reactions of primary nerve deafness (table).

CASE 1.—This patient was born deaf. There was some remnant of hearing, but this gradually disappeared and was all gone by the time the patient reached adult life. Along with this congenital defect in the inner ear, she had three branchial fistulas, one in front of each ear and one in the left side of the neck.

CASE 2.—This patient was born partly deaf, and his remnant of hearing had died out by the age of 10.

CASE 3.—This patient became totally deaf from pneumonia at 6 months of age. The cause of his deafness was a toxic neuritis of the eighth nerve, such as causes a majority of cases of profound acquired deafness in childhood.

CASE 4.—The patient lost part of his hearing at the age of 2 during a severe attack of diphtheria. His two sisters had diphtheria at the same time. All three children were given up by the old family physician, but a young physician who had some of the new diphtheria antitoxin was called in and all three children recovered. However, all three had a permanent, partial deafness, a toxic neuritis of the eighth nerve from the severe general infection. This defect was not progressive. The table shows that the mother had progressive deafness beginning insidiously at 25, apparently otosclerosis. While all three children were deaf, not one had otosclerosis.

CASE 5.—The patient began to lose his hearing at the age of 11, and this loss was progressive. About the age of 25, being a lover of music, he noticed that he was losing the high notes, so that he finally had to give up listening to sopranos on his victrola. At the age of 35 he suddenly became totally deaf one afternoon, about 3 o'clock. There was no vertigo or other subjective sensation except that he was suddenly stone deaf. This sudden loss is sometimes seen in otosclerosis. Examination of the drum membranes of this patient revealed a slight pinkish glow from the promontory on the left. This, with the history of progressive nerve deafness, suggests the labyrinthine type of otosclerosis producing primary nerve deafness. There was no family history of otosclerosis. His mother was slightly deaf, but only after the age of 90, evidently as a result of senile nerve degeneration.

CASES 6, 7 and 8.—The patients had primary nerve deafness for which no cause could be found.

CASE 9.—This patient had a chancre of the tonsil at the age of 16, which was treated only by drops given by mouth. At the age of 43 he suddenly lost his hearing. There was partial recovery after a month, but three months later he suddenly became totally deaf and has remained stone deaf since. This rapid, complete loss of hearing suggests syphilis.

CASES 10 and 11.—The patients had senile nerve deafness.

The remaining 54 patients all showed the tuning fork reactions of conduction deafness, with negative Rinne tests, increased bone conduction and elevation of the lower tone limit. Most of them also showed more or less secondary degeneration of the nerve as evidenced by loss of hearing for the high tones.

Summary of 65 Cases

Case	Age at Onset	Family History of Deafness	Previous Otic Disease	Onset and Course of Deafness	Tympanic Membranes	Diagnosis
1	Birth	None	None	Born partly deaf; remnant of hearing lost by adult life	Right markedly thickened and retracted with loss of luster Left same Has 3 brachial fistulas	Congenital deafness
2	Birth	None	None	Born totally deaf in right ear, partly deaf in left; remnant lost by age of 10	Right slightly thickened with mottling Left normal	Congenital deafness
3	6 mo.	None	None	Acute onset of total deafness with pneumonia	Right slightly thickened with mottling Left normal	Toxic neuritis of eighth nerve from acute infection
4	2	Mother severe, insidious onset at 25, progressive Maternal uncle, one ear only 2 sisters, moderate, since childhood, following diphtheria, not progessive	None	Acute onset of partial deafness with severe diphtheria; not progressive	Right slightly thickened with slight loss of luster Left moderately thickened with loss of luster	Toxic neuritis of eighth nerve from acute infection
5	11	Mother slightly deaf, onset at 90	None	Insidious onset following measles and whooping cough; progressive with loss of high notes first; sudden, total deafness at 35, at 3 p. m.	Right normal Left normal with suggestion of pinkish glow	Primary nerve deafness, cause unknown; otosclerosis possible
6	12	Mother slightly deaf, onset at 60	None	Acute onset of partial deafness with some subsequent improvement and several similar attacks since; not progressive	Right moderately thickened Left thickened with a small healed perforation	Primary nerve deafness, cause unknown
7	35	None	None	Insidious onset, progressive	Right moderately thickened and retracted; diminished luster Left slightly thickened	Primary nerve deafness, cause unknown
8	36	Maternal cousin deaf since scarlet fever at age of 8	Otitis media on right for one week at age of 10 but followed by normal hearing	Insidious onset of deafness during an attack of iritis for which no focus was found	Right slightly thickened Left slightly thickened	Primary nerve deafness, cause unknown (probably same etiology as iritis)
9	43	None	None	Acute onset of severe deafness during a cold with partial recovery; three months later total deafness; chancre of tonsil at 16 treated by drops	Right thickened, lusterless, with calcareous deposits posteriorly Left scarred with marked atrophy and relaxation; moderate retraction	Primary nerve deafness, probably syphilitic

10	53	Maternal cousin deaf *Sister, insidious onset at 25, progressive, uses electrical hearing device	None	At 19 noticed poor hearing in left ear, not progressive; insidious onset in right ear at 53, progressive	Right thickened, retracted, loss of luster Left slightly thickened, retracted, diminished luster	Primary nerve deafness of right ear (senile); tubotympanic conduction deafness of left ear
11	68	Father slightly deaf, onset at 65 Maternal uncle deaf, onset at 70	None	Insidious onset, progressive	Right somewhat thickened with loss of luster Left thickened and scarred with loss of luster and slight retraction	Primary nerve deafness (senile)
12	7	Mother slightly deaf, onset at 60 Half-brother deaf	Chronic otitis media on right for years; no trouble on left	Deaf since onset of otic trouble as a child; progressive since age of 55	Right large, dry posterior marginal perforation, epithelialized Left moderately thick and retracted	Exhausted chronic suppurative otitis media; superimposed senile nerve deafness
13	8	None	Otitis media on left for one year at age of 8	Deaf since discharge from ear at 8; not progressive	Right retracted with calcareous deposits Left retracted, slightly thickened, loss of luster, calcareous deposits	Tubotympanic
14	12	Paternal grandmother deaf, onset at 55 Maternal grandfather deaf since scarlet fever at 20 Maternal aunt deaf since 30, not progressive	Many earaches as a child; bilateral acute otitis media at age of 12	Deaf since otitis media at 12, not progressive in left ear, slightly progressive in right ear	Right absent with cholesteatoma in attic, slight discharge Left moderately retracted, well developed atrophy	Active chronic suppurative otitis media
15	18	Mother very deaf, insidious onset at 50, progressive *Brother deaf, insidious onset at 30, progressive *Maternal aunt deaf, insidious onset at 35 Maternal uncle deaf, insidious onset at 50	Occasional discharge from both ears for years; heard better with discharge from ears	Sudden deafness in left ear at 18 after a deep dive; insidious onset in right ear at 35, progressive	Right absent with dry, epithelialized middle ear Left absent with moist cholesteatoma	Active chronic suppurative otitis media; possible superimposed otosclerosis
16	2	Father slightly deaf, onset at 70 Brother; onset in childhood; went to school for deaf	None	Onset with diphtheria of slight defect in left ear, total deafness in right; at 25 with a head cold, hearing in left ear became worse; not progressive	Right slight mottling Left very slightly thickened, with partial loss of luster	History suggests tubotympanic, but profound conduction defect with slight changes in the membranes indicates possible otosclerosis
17	45	Mother moderately deaf, onset not known	Earaches as a child but no deafness; otitis media on left at age of 53; did not increase deafness	Acute loss in left ear without apparent cause; gradual loss in right ear since then	Right somewhat thickened, with loss of luster Left same	Tubotympanic; possible otosclerosis
18	49	Brother deaf, onset at 50	Recurring otitis media on left ever since measles as a child; otitis media on right two months ago	Mastoidectomy on left ear twenty years ago with complete loss of hearing; gradual loss in right ear since 55	Right slightly thickened and retracted; cholesteatoma of external canal Left, radical mastoid cavity	Tubotympanic with advanced superimposed senile nerve degeneration

* Deaf relative whose history suggests otosclerosis (i. e., progressive deafness beginning insidiously in early or middle adult life).

Summary of 65 Cases—Continued

Case	Age at Onset	Family History of Deafness	Previous Otic Disease	Onset and Course of Deafness	Tympanic Membranes	Diagnosis
19	30	None	None	Otitis media on left at 30; deaf in that ear since; acute loss of hearing in right ear following a severe abdominal operation at 47; not progressive	Right considerably thickened, normal luster Left slightly thickened	Tubotympanic
20	18	Maternal grandmother deaf in one ear only *Maternal cousin, onset at 24, progressive	Earaches and discharge as a child but none after 7 years, with normal hearing	Insidious onset, progressive	Right moderately thickened with moderate retraction Left markedly thickened and retracted with loss of luster	Tubotympanic; possible otosclerosis
21	10	Paternal half-aunt, onset at 45; slightly deaf *Mother, severe, insidious onset at 31, progressive; mother had blue sclerae and brittle bones	Recurring earaches as a child; none after age of 5 and no deafness	Insidious onset, progressive	Right slightly mottled with loss of luster; pink glow from promontory Left slightly mottled	Otosclerosis; fragilitas ossium with blue sclerae
22	12	Paternal cousin deaf in one ear since otitis media as a child Father slight, onset at 69	None	Insidious onset, progressive	Right slight thickening with slight loss of luster Left slight thickening; normal luster	Otosclerosis probable
23	13	Father slightly deaf since a young man; progressive after 65	None	Insidious onset, progressive	Right normal except for very slight thickening; pink glow from promontory Left same	Otosclerosis
24	14	*Mother deaf, onset at 42, progressive *Maternal grandfather deaf *3 maternal aunts deaf, onset in middle life *Sister deaf, onset at 12, progressive *Sister deaf, onset at 30, progressive	Occasional earache as a child; no deafness	Insidious onset, progressive	Right normal except for slight thickening along inferior border Left slightly thickened, slight retraction with loss of luster	Otosclerosis
25	17	*Father, slight, onset at 36, progressive *Mother, severe, onset at 8, progressive *Maternal aunt, progressive deafness *4 sisters, all progressive deafness (father and mother fifth cousins)	None	Insidious onset, not much progression until after 25	Right moderate thickening, loss of luster Left moderate thickening, normal luster	Otosclerosis
26	18	*Father, onset in middle life, progressive Maternal grandfather deaf, onset in old age	None	Total deafness in left ear since brain fever at 18; insidious onset in right ear at 50, progressive	Right moderately thickened with loss of luster Left normal	Probable otosclerosis, right ear; toxic neuritis of left ear from meningitis

27	21	Father, onset at 56 Paternal aunt very deaf following pregnancy Maternal grandmother slightly deaf, onset at 72	Occasional earache as a child, no deafness	Insidious onset, progressive	Right normal except for slight mottling and relaxation; pink glow from promontory Left, slight mottling and thickening with loss of luster	Otosclerosis
28	22	None	None	Acute onset in left ear with a cold; insidious onset in right ear a year later, progressive	Right, very slight mottling and retraction; pink glow from promontory Left moderately thickened with loss of luster	Otosclerosis
29	24	Maternal cousin, progressive, onset at 50 *Maternal cousin, progressive, onset at 30; uses electrical hearing aid *Maternal niece, onset at 7, progressive Maternal niece, onset at birth	None	Acute onset of tinnitus followed a year later by insidious onset of deafness, progressive	Right very slightly thickened Left same	Otosclerosis
30	25	*Father very deaf, onset at 30, progressive; used piece of glazed paper between teeth to hear	Otitis media on left at 38; earache once as a child	Insidious onset, progressive, especially after each of two attacks of influenza	Right moderately thickened; no loss of luster Left very slightly thickened	Otosclerosis
31	25	*Paternal aunt moderately deaf	None	Insidious onset, progressive	Right, slightly thickened Left slightly thickened with loss of luster	Otosclerosis
32	29	*Mother deaf, onset in middle life *Maternal aunt, same	Earaches as a child; no deafness	Insidious onset, progressive	Right, thin scar covers large central perforation Left slight thickening with slight loss of luster	Otosclerosis
33	30	Brother, slight, onset at 30	None	Insidious onset, progressive	Right normal except for slight mottling in posterior superior segment Left diffuse mottling	Otosclerosis
34	30	None	None	Insidious onset, progressive	Right slightly thickened Left slightly thickened with loss of luster	Otosclerosis
35	30	*Mother, onset in middle life, progressive *5 maternal uncles, same *Sister, same	Earaches as a child; no deafness	Insidious onset, progressive	Right slightly thickened and slightly retracted Left moderately thickened and slightly retracted	Otosclerosis
36	33	*Maternal aunt, onset at 40, progressive; uses electrical hearing aid	Earaches every winter as a child; no discharge	Insidious onset, progressive	Right moderately thickened, slight loss of luster Left slightly thickened	Otosclerosis
37	40	?*Paternal aunt very deaf, onset probably in middle life Mother deaf in one ear only	Earaches and occasional discharge as a child; no deafness	Insidious onset, progressive	Right slightly thickened with mottling and slight retraction Left moderately thickened with loss of luster	Probable otosclerosis

Summary of 65 Cases—Continued

Case	Age at Onset	Family History of Deafness	Previous Otic Disease	Onset and Course of Deafness	Tympanic Membranes	Diagnosis
38	Small child	*Mother, father, 4 sisters all with progressive deafness	Many severe earaches as a child; no discharge	Has had some deafness as long as she remembers; progressive since 22	Right normal with distinct pink glow from promontory Left normal	Otosclerosis
39	8	*Paternal grandfather, onset at 16 ?*Paternal great-uncle deaf *Paternal cousin, onset at 12 Father deaf, one ear only, onset at 55; found to be primary nerve (senile) Mother slightly deaf, one ear only, following bad cold at 19, not progressive; found to be tubotympanic ?*Maternal great-grandfather deaf Sister slightly deaf, one ear only	Shooting pains in left ear for ten years	Insidious onset, progressive	Right normal Left normal	Otosclerosis
40	8	?*Father, paternal grandfather and 2 paternal aunts were all deaf, onset not known	None	First noticed at age of 8; does not think it has been progressive (is now very deaf at 60)	Right normal Left normal	Otosclerosis
41	8	*Sister moderately deaf, onset in middle life; uses an ear phone *5 daughters have progressive deafness	None	Insidious onset at 8, progressive, particularly after 45	Right normal Left normal except for loss of luster	Otosclerosis
42	10	Maternal grandfather, onset at 75	Otitis media on left as a child; no residual deafenss	Insidious onset, progressive	Right normal Left normal except for scar in inferior segment	Otosclerosis
43	12	*Mother, onset at 30, progressive Brother, onset at 50, slightly deaf	None	Insidious onset, progressive	Right normal Left normal	Otosclerosis
44	13	?*Paternal uncle had progressive deafness	None	Insidious onset, progressive	Right, thin scar covers large posterior and small anterior perforation Left normal except for relaxation posteriorly; suggestion of pinkish glow	Otosclerosis
45	13	*Paternal grandmother, onset at 40 *3 paternal uncles, onset in middle age	Chronic otitis media on right following scarlet fever at 3 until about 13	Deaf in right ear since 3; insidious onset in left ear at 13, progressive	Right absent with an epithelialized attic perforation Left normal; very marked pink glow from promontory	Otosclerosis of left ear; exhausted chronic otitis media on right

46	13	Mother deaf in one ear only, not progressive 2 maternal cousins deaf *2 sisters, onset at 13, progressive	None	Insidious onset, progressive	Right very slight mottling Left normal	Otosclerosis
47	14	*Mother, father, 4 sisters all with progressive deafness	None	Slight defect first noticed following measles at 14; rapid progression for six months at 30, not much since (now 41)	Right normal; slight pink glow from promontory Left same	Otosclerosis
48	30	*Identical twin sister of patient 47	Several earaches in left ear at 34	Slight defect in left ear noticed at 30; has not progressed in last six years; right ear seems normal (C_1 moderately shortened, Rinne shortened positive)	Right normal Left normal	Otosclerosis
49	15	*Maternal great-grandmother deaf *Maternal grandmother, onset at 30, progressive *2 maternal cousins, onset at 18, progressive *1 uncle, onset at 35, progressive *2 maternal aunts, onset at 35, progressive	Occasional earaches as a child	Insidious onset, progressive	Right normal, slight pink glow from promontory Left same	Otosclerosis
50	16	None	Quite deaf at 14 following typhoid, but this completely cleared up before 16	Insidious onset, progressive	Right normal Left slightly thickened	Otosclerosis
51	17	None	None	Insidious onset, progressive	Right normal Left normal	Otosclerosis
52	18	*Father deaf, progressive *Paternal great-uncle deaf Many other relatives are deaf (was included in genealogy studies published in Archives)	Struck head at 12 with bleeding from ear but no deafness	Insidious onset, progressive	Right slightly thickened, moderately retracted with slight loss of luster Left normal except for slight relaxation	Otosclerosis
53	20	Mother, onset at 86	None	Insidious onset, progressive	Right normal Left normal	Otosclerosis
54	20	*Father, onset at 40, progressive	Recurrent otitis media on right as a child, but without deafness until 20	Defect in right ear noticed about 20; insidious onset in left ear and progressive since 30	Right moderately thickened and slightly retracted, with slight loss of luster Left normal	Otosclerosis on left; tubotympanic on right
55	22	*Father, onset at 40, progressive *2 paternal uncles deaf Mother, onset at 90	None	Insidious onset, progressive	Right normal Left normal	Otosclerosis

Summary of 65 Cases—Continued

Case	Age at Onset	Family History of Deafness	Previous Otic Disease	Onset and Course of Deafness	Tympanic Membranes	Diagnosis
56	24	*Maternal great-grandmother deaf *Grandmother, onset at 40 *Mother, onset at 15, progressive *Brother, onset at 22, progressive *Sister, onset at 36, progressive	None	Acute onset following a cold; progressive	Right normal, with pink glow from promontory Left normal	Otosclerosis
57	25	*Paternal aunt, onset at 30	None	Insidious onset, progressive	Right normal Left normal	Otosclerosis
58	25	*Maternal uncle, onset at 50 *2 great-aunts, onset early in adult life *Son of one of these aunts, onset at 25 *1 sister, onset at 15 *1 sister, onset at 25 *1 brother, onset at 30	None	Insidious onset, progressive	Right normal Left normal	Otosclerosis
59	26	*Father's half-sister, onset at 31, progressive	None	Insidious onset, progressive, until 34; not much change since (now 43)	Right normal Left normal	Otosclerosis
60	28	Father, slight, onset at 65 Mother deaf in one ear only *Maternal grandfather, onset at 28, progressive *Maternal aunt, onset at 28, progressive *2 maternal second-aunts, onset at 30, progressive Maternal great-uncle deaf in old age Maternal second-niece born deaf *Maternal great-uncle, progressive	Earaches as a child; no deafness	Insidious onset, progressive	Right normal, slight relaxation Left same	Otosclerosis
61	30	*Mother, onset at 40, progressive *3 maternal aunts, onset in middle life *Maternal grandmother deaf *Sister, onset at 12, progressive *Sister, onset at 14, progressive	None	Insidious onset, progressive	Right normal Left normal	Otosclerosis
62	30	Grandfather, onset in old age	None	Insidious onset, progressive especially after 50	Right normal Left normal	Otosclerosis
63	30	None	None	Insidious onset, progressive	Right normal Left normal	Otosclerosis
64	38	*Mother, onset at 30, progressive *Sister, onset at 45, progressive *Sister, onset at 62	Earaches as a child; no deafness	Insidious onset, progressive (has chronic sinusitis)	Right normal except for slight atrophy Left normal	Otosclerosis
65	40	Father, onset at 60 *Maternal grandmother, onset at 45 *Maternal uncle, onset in middle life	None	Acute onset of defect with influenza followed by recovery; insidious onset of progressive deafness soon afterward	Right normal Left normal	Otosclerosis (toxic neuritis from influenza cleared up before onset)

CASE 12.—The patient had been deaf since the onset of chronic otitis media in childhood. The table shows that the deafness was not progressive until the patient was 55 years old, when a senile nerve deafness began to be superimposed.

CASE 13.—This patient had been deaf since a discharge from the ear at the age of 8. Both drum membranes showed definite changes. The table shows that here, also, the deafness was not progressive.

CASE 14.—In this case the patient had been deaf since a bilateral otitis media at 12 years of age. The defect had not been progressive except in one ear, and this ear proved to have an active cholesteatoma in the attic. One should notice the family history of this patient. The grandmother evidently had senile nerve deafness, and the grandfather, toxic nerve deafness following scarlet fever; the aunt was deaf at the age of 30, from an unknown cause, but the deafness was not progressive. There is a strong family history of deafness but it is not a history of otosclerosis, nor did the patient have otosclerosis.

CASE 15.—The patient lost his hearing in one ear at the age of 18 after a deep dive, and had increasing deafness in the other ear with recurring discharges from the ears. Active chronic suppurative otitis media may cause progressive deafness, but in an exhausted dry ear the defect is stationary until senile nerve changes supervene. There is, however, a family history of otosclerosis in this patient, and a superimposed otosclerosis is a possible cause for his progressive deafness.

CASES 16 to 20.—These were similar cases of tubotympanic middle ear conduction deafness.

CASE 21.—This case is interesting. The patient had earaches as a baby, but her hearing had been normal until, at the age of 10, there was an insidious onset of progressive deafness. Both drum membranes showed slight changes of tubotympanic disease, apparently left over from the earaches in infancy. On examining this patient, it was noticed that the sclerae of her eyes were deep blue. She was asked whether she ever had broken bones. "Oh, yes, doctor," she replied, "I forgot to tell you. I've always had very brittle bones that break with the least injury and my mother had the same thing, too, and the whites of her eyes were blue like mine." The patient's mother had also had progressive deafness. Here we have an instance of otosclerosis in mother and daughter occurring with fragilitas ossium and blue sclerae. Incidentally, the distinct pink glow from the promontory pathognomonic of otosclerosis was seen shining through the right drum membrane in this patient.

CASE 22.—This patient began to notice the insidious onset of progressive deafness at the age of 12. Both drum membranes showed slight tubotympanic changes. Nevertheless, the diagnosis of probable otosclerosis was made because of the profound conduction defect with the history of progressive deafness in the absence of any acute symptoms of tubal occlusion or of otitis media.

We realize that the diagnosis of otosclerosis in this case is open to question. Fraser of Edinburgh stated that patients suffering from progressive conduction deafness whose drum heads show the result of former inflammation of the middle ear have otosclerosis. In the absence of otosclerosis, deafness is not progressive in such cases. It has been our experience, also, that in the absence of acute exacerbations caused by tubal occlusion with colds in the head, chronic catarrhal otitis media is not progressive.

CASE 23.—This is a similar instance of progressive deafness which started insidiously at the age of 13 and showed slight changes in the drum membranes. The pink glow from the promontory confirmed the diagnosis of ostosclerosis.

CASE 24.—This patient had a strong family history of otosclerosis, her mother, grandmother, three aunts and two sisters all suffering from progressive deafness which started in early or middle adult life. She first noticed difficulty in hearing at the age of 14, and her deafness had been progressive. Because of this and of the profound conduction defect, the diagnosis of otosclerosis was made in spite of the slight changes in the drum membrane.

CASES 25 to 37.—These patients had progressive deafness in the presence of slight changes in the drum membranes, but the condition was diagnosed as otosclerosis.

Altogether, there were 17 cases diagnosed as otosclerosis in spite of the slight changes in the drum membranes. Of the 17 patients, 11 gave a family history of otosclerosis, and 4 showed the pink glow from the region of the promontory. By comparison, only 2 of the 11 patients with primary nerve deafness and 2 of the 9 patients with tubotympanic middle ear deafness gave a family history of otosclerosis, and none showed the pink glow from the promontory.

The remaining 28 patients showed one or both tympanic membranes entirely normal, and all showed the well known Bezold triad of conduction deafness. These cases were all diagnosed as cases of otosclerosis. Six of them showed the pink glow from the promontory. Twenty-two of the 28 patients gave a family history of otosclerosis. One family was of particular interest.

CASE 41.—This patient had a sister with progressive deafness, and she herself had typical otosclerosis. She married her fifth cousin. Progressive deafness developed in her husband at about 40 years of age, but he died in his fifties before his defect had become very severe. They had five daughters, all with progressive deafness, and we were able to examine four of them, as follows:

One patient (case 25) had progressive deafness, and the condition was diagnosed as otosclerosis in spite of moderate thickening of both drum membranes.

Another daughter (case 38) had typical otosclerosis with the pathognomonic pink glow from the promontory.

The patients in cases 47 and 48 were identical twins. The first one had typical otosclerosis with Bezold's triad of conduction deafness in the presence of normal drum membranes, through one of which a pink glow from the promontory was visible. The second one had normal drum membranes and beginning conduction deafness, but the Rinne test was still positive in her good ear. Nevertheless, with the family history, the normal drum membranes and beginning conduction deafness, a definite diagnosis of early otosclerosis was made.

The question will immediately arise, Are we justified in the diagnosis of otosclerosis in all of these cases? Bezold, who first connected the pathologic findings with the clinical picture of otosclerosis and who had a greater opportunity than any of us to confirm his diagnoses by postmortem examination, saw more than 1,000 cases. He obtained a

family history of deafness in less than 52 per cent of his cases. We obtained a family history not only of deafness, but of deafness suggesting otosclerosis, in more than 73 per cent of our cases from the Washington League. Our higher percentage is attributable to the fact that the members of the Washington League were asked to inquire into their family histories before coming to the examination. Other patients with otosclerosis seen in Chicago gave a family history in only 58 per cent of instances, but they had not been forewarned to ascertain their family histories.

Bezold stated that normal or nearly normal findings on the tympanic membrane and the triad of conduction deafness are necessary to diagnose otosclerosis. Our 28 patients with normal drum membranes conform to this criterion. In the cases with slight changes in the drum membranes, the diagnosis is more difficult, but, nevertheless, it can be made, as Fraser stated, from the progressive character of the defect. The clinical histories of all of these patients conform to that of otosclerosis. Nearly all had repeated and prolonged treatments at the hands of one or several aurists, and although at the time of the treatments many believed that they were improved, as time went on they realized that the treatments not only failed to improve the hearing, but had no effect on the progress of the defect.

It should be pointed out that the proportion of cases diagnosed as otosclerosis showing slight changes in the drum membranes is no larger than the proportion of persons with normal hearing who show slight changes in the drum membranes. These changes are residues of tubotympanic processes in childhood, which do not in themselves impair the hearing, as is shown by the fact that the hearing returned to normal in all of these patients before the onset of progressive deafness in early or middle adult life. These residues can no more prevent the development of otosclerosis than they can cause it; therefore, a certain number of cases of otosclerosis will show slight alterations in the drum membranes.

Finally, those who are doing pathologic work on temporal bones in this country, particularly Crowe, Guild and Hagens, find not only that otosclerosis is a very common disease, but that it is present in cases in which it was not suspected during life; it is more common than clinical observation would indicate.

Our findings for the Washington League are summarized in figure 6. Of the 65 cases, there were 11 of primary nerve deafness, 9 of tubotympanic middle ear disease beginning, frequently, in childhood, and 45 of otosclerosis with primary fixation of the stapes. Seventeen of the latter were diagnosed as otosclerosis in spite of slight changes in the drum membranes.

Our figures from the other two groups studied correspond closely to these. There were 60 cases from the Chicago League for the Hard of Hearing: 12 of primary nerve deafness, 6 of tubotympanic middle ear disease, 11 of otosclerosis with slight changes in the drum membranes and 31 of typical otosclerosis with normal drum membranes.

The 40 consecutive cases from our private practice showed similar results, with 7 cases of primary nerve deafness, 5 of tubotympanic middle ear disease, 8 of otosclerosis with slight changes in the drum membranes and 20 of typical otosclerosis with normal drum membranes.

One frequently sees the diagnosis of "combined deafness." How many of our cases showed a combined deafness or a combination of factors responsible for the loss of hearing? Considering the entire group of 165 adults with severe deafness, there were 30 cases of primary nerve deafness. In only 2 was there a combination of etiologic

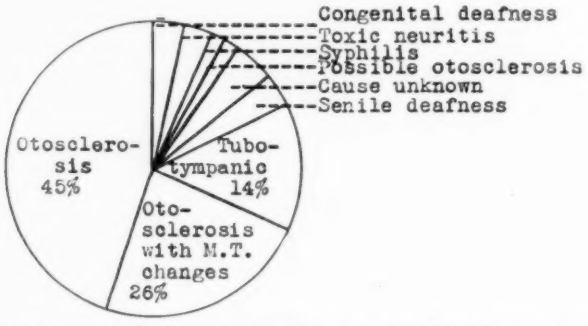

Fig. 6.—Etiology of deafness in members of the Washington League for the Hard of Hearing.

factors. One patient became partially deaf from measles at the age of 3. This defect was not progressive until the age of 16, when she began to work in a very noisy factory; her hearing began to grow worse—a toxic neuritis with a superimposed occupational nerve deafness. The other patient had been moderately deaf in one ear since she was 19, and the deafness had not been progressive. Examination showed a tubotympanic conduction deafness in this ear. At the age of 54 she began to lose her hearing in the other ear, examination of which showed a primary senile nerve deafness.

There were 135 cases of conduction deafness. Nearly all of these showed a certain amount of secondary degeneration of the nerve, because long-standing tubotympanic conduction deafness is always associated with a certain amount of secondary degeneration of the nerve, as is advanced otosclerosis. However, this degeneration is not due to a second etiologic factor except in patients of advanced years, in whom the nerve defect is accentuated by senile changes.

We encountered 3 cases of conduction deafness with combined etiologic factors. One patient had tubotympanic deafness from childhood in one ear with a superimposed otosclerosis involving the other ear. A second patient had a unilateral dead labyrinth from meningitis with superimposed otosclerosis in the opposite ear. The third patient had an exhausted chronic suppurative otitis media in one ear with a superimposed otosclerosis in the opposite ear.

Thus we found a combined etiology in only 5 of the 165 adults examined. The term "combined deafness" as often applied to conduction deafness with secondary degeneration of the nerve is misleading and confusing.

Only 2 cases of chronic sinusitis were found among the 165 deafened adults. One patient had typical otosclerosis with a strong family history and with normal drum membranes. The other had chronic

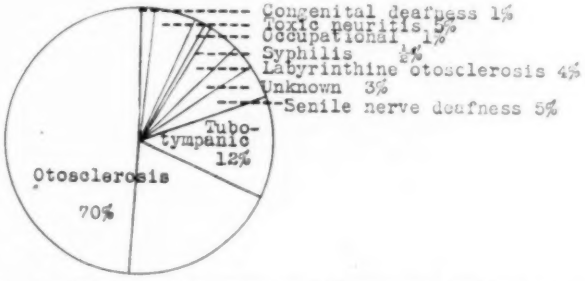

Fig. 7.—Etiology of deafness in 165 severely deafened adults.

suppurative otitis media with cholesteatoma; the chronic sinusitis was a possible contributing factor. One patient with tubotympanic middle ear deafness had atrophic rhinitis with extensive crusting, doubtless the important etiologic factor in her deafness. The remaining 162 deafened adults had no clinical symptoms and no clinical findings of nasal pathologic changes or chronic sinus infection.

A point that seems worth emphasizing is the general health of patients with otosclerosis. The statement has been made that persons with otosclerosis have a constitutional inferiority. We found that almost without exception these people enjoy excellent health and are mentally and physically normal in every respect except for their hearing. We did find 1 case of fragilitas ossium and blue sclerae among the 115 patients with otosclerosis. There was no case of Paget's disease.

SUMMARY

One hundred and sixty-five adults suffering from deafness severe enough to constitute a handicap have been studied. Thirty were

afflicted with primary nerve deafness, caused by a congenital defect in 2, by toxic neuritis from an acute infectious disease in 5, by a noisy occupation in 2 and by senile changes in 7. Syphilis was the probable cause in 1 case. In 12 patients with primary nerve deafness the cause was unknown, but in 6 of these the labyrinthine type of otosclerosis was suspected. Twenty patients suffered from a tubotympanic middle ear form of deafness. One hundred and fifteen patients had conditions diagnosed as otosclerosis with primary fixation of the stapes.

CONCLUSIONS

Our conclusions are that only 12 per cent of severe adult deafness is due to middle ear disease beginning in childhood and is in any way preventable by examination of school children (fig. 7). Severe deafness in adults profound enough to constitute a handicap is due to otosclerosis with ankylosis of the stapes in the majority of instances (70 per cent) and is neither preventable nor amenable to treatment in the present state of knowledge.

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FRACTURES OF THE LARYNX

WITH REPORT OF A CASE

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In reviewing the literature on fractures of the larynx one is struck by the paucity of material, especially that published in more recent years. While the condition is not common, it is not such a rarity as one might be led to believe. Many of these cases are not diagnosed as such, and many more are not reported, thus producing the scarcity of available information.

Hubicat's [1] report published in 1620 is the first to appear in the literature, and it is interesting to note that even at this early date he performed a tracheotomy, using a lead tube for a cannula, which he left in place for three weeks, the patient making a complete recovery. In 1866, Hunt [2] collected a series of 26 cases. Two years later Henoque [3] added 26 more. In 1895, Dunham [4] brought the total to 75 and reported them extensively. The latest review seems to be that made by Hoffman [5] in 1898, when he recorded a total of 140 cases. Since then I have been able to collect 16 more.

ETIOLOGY

Fractures of the larynx are much more common in adults than in children and in men than in women. In all probability this is because the cartilages undergo calcification around the twenty-fifth year, and adults, especially men, are more often exposed to the particular type of trauma producing fractures. Cases are not unknown in children, however, and several have been reported in children of various ages. Hume [6] reported a fracture of the thyroid cartilage in a child of 6 years,

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Read before the Iowa Academy of Ophthalmology and Otolaryngology, Aug. 18, 1932.

1. Hubicat, Nicolas: Surgical Inquiry, quoted by Louis: Bronchotomy, London, Sydenham Society, 1848, p. 229.

2. Hunt, William: Fractures of the Larynx, Am. J. M. Sc. **51**:378 (April) 1866.

3. Henoque: Gaz. hebd. de méd. **5**:610 (Sept. 25); 625 (Oct. 2) 1868.

4. Dunham: Fractures of the Larynx, M. News **67**:208, 1895.

5. Hoffman: Fracture of the Larynx, quoted by Sauer.[7]

6. Hume: Surgical Emphysema: Fracture of the Cricoid Cartilage; Death, Lancet **1**:987, 1882.

and Sauer [7] observed one in a boy of 14. The age at which ossification or calcification occurs is variable; most observers state that the thyroid cartilage begins to calcify prior to the twenty-fifth year, and the cricoid cartilage about five years later. The epiglottis, the cuneiform cartilage and the apexes of the arytenoid cartilages are composed of yellow elastic tissue and show little if any tendency toward calcification. Hunt observed a man 103 years of age whose larynx, post mortem, showed not even beginning ossification.

MECHANISM AND PATHOLOGY

The larynx does not fracture easily because of its great mobility and the protection offered it by the chin. Compression from side to side or from before backward as in throttling is one of the most frequent methods of producing fracture. Escalada [8] stated that from about 55 to 80 Kg. of pressure is necessary to fracture the average adult larynx. Scheier attempted to fracture the thyroid cartilage of a woman 19 years of age by striking it with his closed fist at the time of postmortem examination; after repeated attempts, he was unsuccessful. In 15 other bodies, however, he was able to produce fractures by exactly the same force applied in a similar manner.

Throttling or choking with the hands is one of the most common causes of fracture of the larynx. Hanging practically never produces a fracture of the larynx; some observers say never, but Delavan [9] reported a fracture of the thyroid produced in this manner. The act of stumbling and falling forward, striking the neck and producing compression from before backward is frequently mentioned as the etiologic factor. De Roaldes [10] reported the case of a man who fractured the left cornua of the thyroid cartilage while trying to induce vomiting to dislodge food from his throat. Andrews [11] described the unusual case of an engineer who in the act of opening the throttle on his locomotive was seized by a severe attack of sneezing; he felt a crack in his throat and immediately complained of severe pain in the region of his larynx; three days later a fracture of the thyroid cartilage was found. Hawkes [12] reported the case of a youth of 19 who received a

7. Sauer, W. E.: Fractures of the Larynx, J. Missouri M. A. **9**:83, 1898.

8. Escalada: Surg., Gynec. & Obst. **23**:431, 1916 (Internat. Abstr. Surg.).

9. Delavan, D. B.: Comminuted Fracture of the Larynx: Accidental Tracheotomy; Multiple Trauma; Extensive Frostbite; Recovery, M. Rec. **84**:927, 1913.

10. de Roaldes, A. W.: Report of a Case of Incomplete Fracture of the Larynx; Self Inflicted Violence, New York M. J. **65**:178, 1897.

11. Andrews, James: Fracture of the Larynx, Lancet **1**:648, 1912.

12. Hawes, C. M.: Fracture of the Larynx, West Virginia M. J. **2**:351, 1907-1908.

fracture of the neck when he was struck by a fast-pitched base ball. In Uren's [13] case a fracture of the thyroid cartilage was produced when the patient was suddenly thrown forward in his car, striking his neck against the steering wheel.

Multiple fractures are usually produced by a crushing injury or by severe violence of some nature, such as the dropping of a window on the neck or being caught in an elevator shaft.

Mullen [14] reported a fracture of the thyroid cartilage and both arytenoid cartilages in a man who was caught between an elevator and the floor; both of the arytenoid cartilages were torn loose from their attachments and dislocated anteriorly.

Fractures may involve any or all of the cartilages; they may be simple, compound or comminuted. Because of its prominence, the thyroid cartilage is more frequently involved than any of the others, and fractures of the cricoid cartilage are next in frequency. In Fisher's [15] 75 cases he reported the following incidence:

Thyroid cartilage	29
Cricoid cartilage	11
Thyroid and cricoid cartilages	9
Hyoid bone and thyroid cartilage	3
Hyoid bone and thyroid and cricoid cartilages	2
Hyoid bone, thyroid and cricoid cartilages and trachea	1
Hyoid bone, thyroid cartilage and trachea	3
Trachea and thyroid cartilage	1
Cricoid cartilage and trachea	2
All cartilages	14

Hoffman found the thyroid cartilage fractured in 63 of 124 cases, the cricoid cartilage in 17 and more than one cartilage in the remainder. Henoque reported fracture of the thyroid cartilage in 23 cases, fracture of the cricoid cartilage in 7 cases and fracture of both of these cartilages in 7 cases. In the 16 cases I have tabulated, 10 fractures were of the thyroid cartilage, 1 of the cricoid cartilage, 1 of the thyroid and arytenoid cartilages and 1 of the arytenoid cartilages alone; in the remaining 3 cases the location of the fracture was not mentioned.

SYMPTOMS

The symptoms and signs are variable in all cases, depending on the cartilage involved, the extent of the injury and several other factors. Pain is usually the first symptom, and it is present in practically every

13. Uren, C. T.: Fracture of the Larynx, Ann. Otol., Rhin. & Laryng. **41**:513 (June) 1932.

14. Mullen, T. F.: Fracture of the Larynx, Ann. Surg. **80**:660, 1924.

15. Fisher, quoted by Mullen.[14] (Original article not found.)

case. It is usually quite severe and is accentuated by phonation or deglutition. Tenderness and swelling appear early and become so marked that manipulation of any sort is almost out of the question. Loss of consciousness is not an uncommon occurrence if the injury has been severe. Bassères [16] reported a case in which the unconsciousness persisted for nine hours. Oertel [17] described the case of a colored man who was struck on the larynx by a jack handle, with loss of consciousness for several minutes. Sharpe [18] had a patient in whom unconsciousness appeared immediately after striking his neck on a board. Vitalba [19] reported a case in which there was prolonged inhibition of the respiratory and cardiac centers with loss of consciousness lasting for many hours; the pulse rate remained at 40 for three weeks.

Occasionally dyspnea is absent, but more often it is present in varying degrees of intensity. Several investigators have reported death from asphyxiation before tracheotomy could be performed. The dyspnea may be due to edema of the soft tissues of the neck and the membrane of the larynx or to blood in the trachea. Sudden emphysema producing asphyxiation and death several days after the accident has been reported. Lodgment of a fragment of loose cartilage in the air passages has been responsible for dyspnea in some cases.

Hoarseness or laryngitis is commonly observed; it may appear early or many months after the injury. Morgenthau [20] saw a patient three weeks after injury whose only complaint was hoarseness. Sajous [21] recorded 2 cases in which there were no symptoms at the time of injury, but two years later the 2 patients were seen again with severe laryngitis and almost complete loss of voice. Sajous' explanation in these cases is that the fracture may be incomplete, and if it involves only the upper portion of the thyroid cartilage, sparing the lower part, violent symptoms are usually absent.

Hemoptysis is a common finding in most cases, though it is usually not severe. The blood ordinarily is mixed with air, which gives it a frothy appearance, but some cases of brisk hemorrhage have been reported. McDougall saw a man who spat up 2 teacupfuls of blood following fracture of the thyroid cartilage. Hemoptysis was present in

16. Bassères, quoted by Oertel.[17] (Original reference not found.)

17. Oertel, T. E.: Fracture of the Larynx, South. M. J. **17**:619, 1924.

18. Sharpe, W. S.: Case of Compound Fracture of Larynx: Recovery, J. Laryng. & Otol. **42**:682, 1927.

19. Vitalba, cited by Garel, J., and Gignoux: Fractures of the Larynx, Lyons méd. **129**:969 (Dec. 10) 1920.

20. Morgenthau, G. L.: Fracture of the Thyroid Cartilage, J. A. M. A. **17**: 544 (Oct. 10) 1891.

21. Sajous, C. E.: M. & S. Reporter **48**:63, 1883.

10 of 30 cases reported by Harris.[22] Regardless of the amount of bleeding, it is always a serious complication, usually indicating a tear in the mucous membrane and an avenue for infection. Ecchymosis of the soft tissue of the neck may be seen in cases in which the injury has been severe.

Crepitation can usually be elicited, but should not be taken as a positive sign of fracture; conversely, its absence should not rule it out. Lind [23] said that crepitation is frequently felt in a normal larynx. Mullen described a sensation similar to crepitus produced by the normal larynx gliding over the anterior surface of the cervical vertebrae; this he said should not be confounded with real crepitus. In many cases crepitation is hidden by emphysema. The air may be localized to the tissue in the immediate vicinity of the larynx, or it may involve the entire surface of the body. Middleton [24] reported a case of emphysema in which the pericardial fat and the mediastinum were loaded with air. In Hume's case emphysema was so extensive that compression of the lungs resulted. Hill saw a case in which the emphysema involved the entire surface of the body. Story [25] described vividly an emphysema so extreme that the eyeballs protruded from the sockets.

There is always a possibility of necrosis of the cartilage and other inflammatory reactions in cases due to direct force, and the danger of cellulitis of the neck is ever present. Large cervical abscesses may form, with the persistence of a sinus tract and the formation of sequestrums.

DIAGNOSIS

The diagnosis is not as simple as one might expect; many times, in fact, it is impossible to demonstrate the fracture by any diagnostic procedure. Botterham said that in some cases it is difficult to diagnose accurately the lesions produced by a blow on the larynx, for the only symptom of fracture of cartilage not present in mere contusion is crepitus, and this is not appreciable in all cases.

Emphysema and bloody sputum are probably the most constant signs pointing to fracture; they were present in almost every case that has been reported. Hunt, after reviewing his series of cases, said: "I should regard bloody sputum in conjunction with the other symptoms as almost diagnostic and if the voice should be hoarse, the larynx is almost surely the seat of laceration."

22. Harris, T. J.: Fracture of the Larynx, with Report of a Case, M. News **66**:208, 1895.

23. Lind, S. C.: Fracture of the Larynx, Ohio State M. J. **21**:406, 1925.

24. Middleton, G. S.: A Case of Hæmoptysis and Surgical Emphysema Arising from Fracture of a Necrosed Larynx, Glasgow M. J. **42**:343, 1894.

25. Story, G. B.: A Case of Fracture of the Cricoid Cartilage, M. Rec. **51**: 636, 1897.

A change in the configuration of the neck is usually a diagnostic point of importance. Flattening of the pomum adami is significant, especially if it is associated with an abnormal movement of the cartilage.

As has been mentioned, crepitus may or may not be of value. Its absence does not rule out a fracture, and its presence is not diagnostic.

Roentgenograms taken early may or may not reveal the fracture; pictures taken a few days later, however, will usually demonstrate it very well. In Lind's case a fracture of the thyroid cartilage was definitely diagnosed clinically at the time of injury, but the roentgenologist gave a negative report. Several days later a second picture was taken, and a definite line of fracture was demonstrated.

PROGNOSIS

A fracture of the larynx must always be considered as a serious condition, and the prognosis should always be guarded. It depends largely on the extent of the fracture and the cartilage involved. Multiple and comminuted fractures offer a poorer prognosis than do the simple ones. Involvement of the cricoid cartilage is uniformly more fatal than involvement of the thyroid cartilage. According to the mortality statistics of the early observers, fractures of the larynx offer a poorer prognosis than fractures of the skull. Durham reported 88 per cent mortality in 69 cases. Gürlt [26] placed the mortality at 70 per cent, Fisher at 78 per cent, and Albert at 80 per cent.

More recent statistics prove that the death rate has been greatly overestimated in the past. In 1895, Harris found the mortality to be around 30 per cent, with the fractures distributed as follows: 5 of the cricoid cartilage, with 1 fatality; 5 of the cricoid and thyroid cartilages, with 3 fatalities, 19 of the thyroid cartilage alone, with recovery in 80 per cent. One patient with fracture of the hyoid bone died.

Death may ensue immediately following the injury, from asphyxiation or from hemorrhage; again it may occur days later from infection, pneumonia or sudden emphysema. Hume's patient died suddenly on the third day from rapid expulsion of air into the tissues of the neck. Many patients have a resulting stenosis of the larynx which necessitates operation and possibly a permanent tube. Practically all of these patients have an impairment of the voice during the remainder of their life.

TREATMENT

The treatment may be medical or surgical, depending on the findings in each case. The indications primarily are to relieve the urgent

26. Gürlt: Handbuch der Lehre von den Knochenbruchen, Hamburg, G. Grote, 1862-1864, vol. 2, p. 317.

symptoms, such as hemorrhage and dyspnea; secondly, to avoid sepsis and pneumonia.

Many of the less severe cases warrant medical or palliative treatment. In those in which there is no hemorrhage or sign of asphyxiation, symptomatic measures may be instituted. The patient should be kept in bed under the constant vigilance of a nurse who can recognize the onset of any untoward symptoms. Absolute rest of the voice must be insisted on. Morphine and sedatives are helpful in keeping the patient quiet and in lessening his apprehension. The room should be kept moist by some form of steam producer. An ice collar for the neck is comforting to the patient and also lessens the swelling. If food and water cannot be taken by mouth, proctoclysis should be instituted. The surgeon in charge should be in readiness to perform an immediate tracheotomy if dyspnea suddenly appears.

Many physicians feel that surgical intervention is indicated as soon as a fracture can be diagnosed; they believe that one is taking more or less of a chance in every case in which medical treatment is employed. Solis-Cohen [27] said: "Unless forestalled by precautionary tracheotomy and the insertion of a tube, suffocative phenomena may intervene at any time from edema, displacement of fragments, or occlusion of the caliber of the air tubes with clots. Better far that an unnecessary tracheotomy be performed without detriment to the patient than that a number of cases should be allowed to terminate fatally for the want of it." Hunt recommended tracheotomy when emphysema and bloody sputum are present. Keen [28] advised tracheotomy in all cases of fracture of the larynx as soon as the diagnosis is confirmed. Stimson,[29] in his book on fractures, advised the physician not to wait until the dyspnea has become extreme, for its increase at the last is often so rapid and so sudden that death takes place before relief can be given; therefore, he said that "it is the better part of prudence to interfere early and before the intervention is made actually necessary by defective breathing."

The dyspnea may appear several days after the injury when the patient is apparently on the road to recovery; such a case was reported by Atlee.[30] The child had been perfectly well for three days following the injury, and then quite suddenly emphysema of the whole upper extremity developed and the child died of asphyxiation.

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27. Solis-Cohen, J., in International Encyclopedia of Surgery, edited by John Ashhurst, New York, William Wood & Company, 1884, vol. 5, p. 55.

28. Keen, W. W.: Surgery: Its Principles and Practice, Philadelphia, W. B. Saunders Company, 1908, vol. 2, p. 154; vol. 3, p. 470.

29. Stimson, L. A.: A Practical Treatise on Fractures and Dislocations, Philadelphia, Lea Brothers & Co., 1899, vol. 1, p. 298.

30. Atlee: Fractures of the Larynx, Am. J. M. Sc. **35**:121, 1858.

In many cases tracheotomy is difficult to perform because of the emphysema, swelling and extravasation of blood. Jackson [31] advised low tracheotomy in all cases in which it is possible in order to lessen the possibility of tracheal stenosis. If the tube is to be left in place only a short time, it is not so important, but if it is to remain in place for weeks, he believes that it should be removed and placed in the low position even if it necessitates a second operation.

In certain cases tracheotomy may be supplanted by laryngofissure. If there should be marked displacement of the cartilages, this procedure allows reduction more easily than does tracheotomy. It also permits suturing of lacerations and the control of hemorrhage. Suturing of the cartilages as yet has not proved satisfactory; proper position can be maintained best by packing around the tube, which is left in place for an airway. The wound should be left open and the dressings changed frequently to lessen the possibility of infection. Uren immobilized the larynx in his case by splinting with pieces of tongue blade about 2½ inches (6.27 cm.) long covered with gauze bandage. These were placed perpendicularly along the sides of the larynx and held in place by adhesive straps; perfect union was obtained.

In cases in which stenosis seems certain or in which it has already occurred, laryngotomy as performed by Killian offers the best means of restoring the normal lumen. He divides the thyroid cartilage in the midline, including, if necessary, the cricoid cartilage both anteriorly and posteriorly. All scar tissue is then removed with scissors, after which the skin is stitched to the mucous membrane. A soft rubber drain is then inserted into the larynx and attached to the tracheotomy tube to hold it in place. The wound is packed open, and the dressings are changed daily; each time a slightly larger tube replaces the smaller one, until the desired opening is maintained. The tube stimulates the absorption of scar tissue and the epidermidalization of the interior of the larynx; when these processes are complete, the tube is removed and the wound closed.

REPORT OF A CASE

During a boxing match the patient received a hard blow on the left side of his neck which dazed him for a short time. He did not become unconscious and was able to finish the contest. In a few hours the pain became severe; his neck became swollen and he experienced some difficulty in breathing. He thought that the sputum was blood-tinged, but was not certain. A few hours after the injury the swelling increased, and his voice was almost a whisper. He was referred to me about three days later because of hoarseness.

Examination revealed a well developed white man about 30 years of age, who apparently was suffering from pain and difficulty in breathing. Questioning at this

31. Jackson, Chevalier: High Tracheotomy and Other Errors the Chief Causes of Chronic Laryngeal Stenosis, Surg., Gynec. & Obst. **32**:392, 1921.

time was difficult, but he said that he had had extreme dysphagia; he had not eaten for three days. His neck was swollen to such an extent that it was flush with the lower jaw on each side. The pomum adami was not visible and was scarcely palpable. Any manipulation was extremely painful. The swelling was firm and boardlike in consistency, and no crepitus was elicited.

Larynogoscopic examination showed considerable swelling in the arytenoid region, and the epiglottis was so edematous as almost to hide the true cords. There seemed to be some limitation of motion on the left side. No other pathologic changes were noted. The temperature was 101 F.; the pulse rate, 88, and the respiratory rate, 24.

Roentgenograms taken at this time did not reveal any line of fracture. In spite of this, a diagnosis of fracture of the thyroid cartilage and cellulitis of the neck was made.

The patient was sent to the hospital, where he was confined to bed and placed in a steam tent. Hot moist packs were applied almost constantly to his neck. He was placed on a liquid diet, and opiates were given as necessary. The nurses were instructed to watch for signs of asphyxiation or exhaustion, and a tracheotomy set was kept in readiness.

Two days later an area of softening appeared in the right side of the neck, and an incision into this evacuated about 1 ounce (30 cc.) of thick, creamy, yellow pus. Following this the dyspnea and the dysphagia improved. The wound continued to drain, and about three weeks later a small piece of what appeared to be calcified cartilage was extruded. The discharge lessened but never ceased entirely, and after several weeks a definite sinus tract was formed. The hoarseness continued to get worse until it was almost impossible for the patient to whisper.

At this time laryngoscopic examination showed almost complete loss of motion in the left vocal cord and a large mass of granulation tissue in the anterior commissure, evidently where the sinus communicated with the interior of the larynx. Operation was suggested for removal of sequestrums, and was performed by Dr. D. M. Lierle at the University Hospital. He made the following report: Under procaine hydrochloride anesthesia a straight incision was made from the lower border of the hyoid bone to the inferior border of the cricoid cartilage. There was an infected tract lined with granulation tissue which extended from the lower border of the cricoid cartilage to the upper border of the thyroid cartilage. At this point four sequestrums of cartilage were found, and practically the entire wall of the thyroid was necrotic on each side. The sinus tract led into the anterior commissure. The thyroid was curetted and the sequestrums removed. A tracheotomy tube was left in place and the wound closed with drainage.

The patient made an uneventful recovery; the wound closed rapidly, and the granulations in the anterior commissure disappeared without further treatment. The quality of the voice improved to a great extent, but never returned to normal.

CONCLUSIONS

1. Fractures of the larynx are not common because of the mobility and the protected position of the larynx.

2. Diagnosis is difficult in many cases, and roentgenograms are of little value.

3. The mortality is high, probably in the neighborhood of 30 per cent.

4. Palliative treatment may be warranted occasionally, but it is far safer to perform tracheotomy in all cases.

5. Laryngofissure and laryngotomy may be necessary in some cases.

SUPPURATION OF THE PETROSAL PYRAMID

ROENTGENOLOGIC PROBLEMS

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Structurally, the petrous portion of the temporal bone may be pneumatic or diploic. Either type can be recognized on the roentgenogram. As the pneumatic petrous pyramid is the one under discussion, the title of this symposium [1] might properly be "Suppuration of the Pneumatic Petrous Pyramid."

"Petrositis," "apicitis" and "suppuration of the petrous pyramid" are general terms and are applicable to an infection of a petrous pyramid of any structural type. These terms should not be applied to the clinical entity which Dr. Kopetzky described, except in a general way. The proper designation for the lesion in question is "acute coalescent petrositis" or "empyema of the petrous apex with inadequate drainage."

Roentgenographically, the petrous pyramid in any acute or chronic otitic infection shows a change from the normal—either a diminution in aeration due to congestion of the membrane lining the air cells or an increase in density due to productive osteitis. These changes do not indicate the presence of an acute coalescent petrositis for which immediate surgical intervention is imperative.

It is known that in the presence of acute otitis media the roentgenogram of a pneumatized mastoid process shows a slight diminution in aeration. This is due not to an infection in the mastoid process, but to congestion and edema of the cell-lining membrane which are secondary to and accompany the otitic infection. Likewise the roentgenogram of a pneumatized petrous pyramid, in the presence of acute otitis media (fig. 1), shows a slight diminution in aeration which is due to the congestion and edema of the lining membrane. This is not due to an infection in the pyramid.

The petrous pyramid shows a diminution in aeration (fig. 2) in the presence of acute coalescent or destructive mastoiditis, even though

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From the Department of Roentgenology, Beth Israel Hospital, Dr. I. S. Hirsch, Director.

Read at the thirty-ninth annual meeting of the American Laryngological, Rhinological & Otological Society, Chicago, June 10, 1933.

1. Symposium on Suppuration of the Petrosal Pyramid, presented at the meeting of the American Laryngological, Rhinological and Otological Society, June 10, 1933. It includes this paper and one by S. J. Kopetzky (Problems Concerned with Empyema of the Petrous Apex, Arch. Otolaryng. **18**:47 [July] 1933).

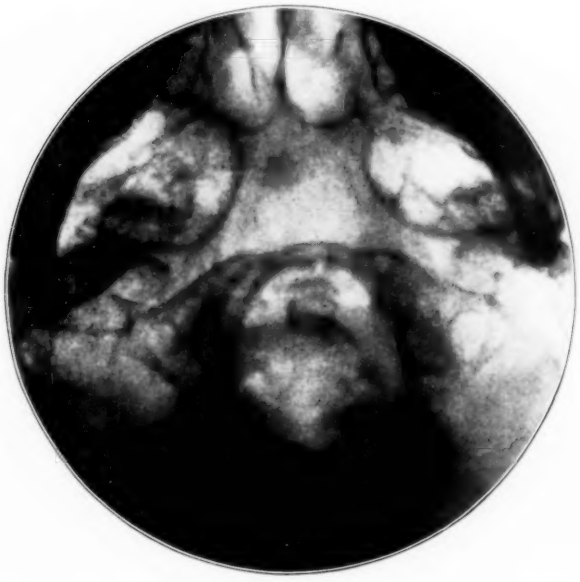

Fig. 1.—March 20, 1933. Clinical diagnosis: acute otitis media. The right petrous pyramid shows a slight diminution in aeration.

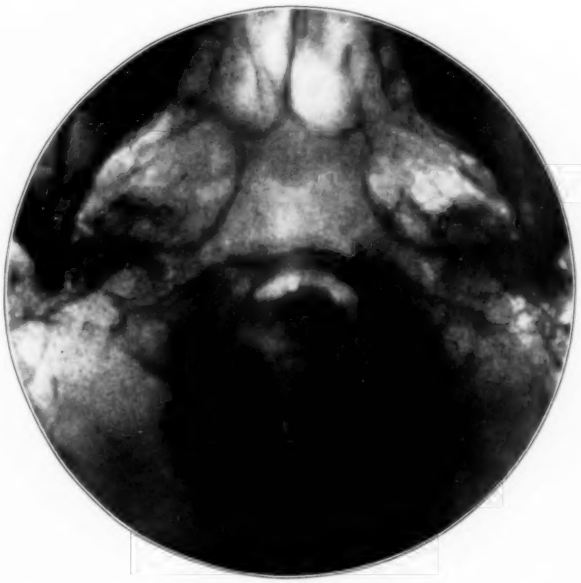

Fig. 2.—Same case as in figure 1. April 5, 1933. Clinical diagnosis: destructive mastoiditis. The right petrous pyramid shows a diminution in aeration. There are no destructive changes in the petrous apex.

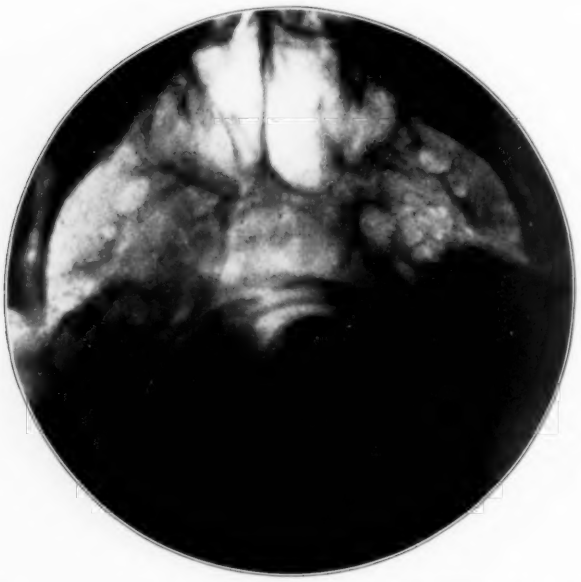

Fig. 3.—Same case as in figures 1 and 2. April 24, 1933. Mastoidectomy was performed on April 10, 1933. A diagnosis of complicating empyema of the petrous apex was made on April 24, 1933. The petrous apex shows halisteresis with destructive changes.

Fig. 4.—The left petrous apex shows halisteresis and destructive changes. The patient is symptom-free except for otorrhea.

there is no extension of the infection into the pyramid. This diminution in aeration is proportionate to the congestion and edema of the membrane lining the air spaces.

Specifically, the petrosa shows essentially the same roentgen findings in acute otitis media as in acute coalescent mastoiditis.

From an anatomic standpoint, it is possible for an infection to extend into the petrous apex and for petrositis to follow as a complication of otitis media or of destructive mastoiditis before surgical treatment has been instituted. Our experience has been limited to cases in which mastoidectomy had been performed.

The observations that follow are based on an analysis of twenty cases of infection of pneumatic petrous pyramids with suppuration. These cases were divided into three groups: acute, subacute and chronic.

1. Acute cases, requiring immediate surgical intervention for drainage of the petrous apex. These cases present a definite symptom complex, described by Kopetzky and Almour, which is due to empyema of the petrous apex with inadequate drainage. In brief, there is a history of mastoidectomy on a well pneumatized bone, followed by an apparently normal convalescence with a period of well-being and then a sudden reappearance of the aural discharge, a low grade sepsis and pain along the distribution of the ophthalmic branch of the fifth nerve, usually referred to as orbital or retro-orbital in location.

Roentgen examination [2] of acute coalescent petrositis with inadequate drainage shows a diminution in the aeration of the pyramid (fig. 3), which is particularly marked in the basal portion. The trabeculae in the apex are thin and decalcified. As the lesion progresses, the halisteresis becomes more marked. This is followed by destructive changes. The decalcification may be so intense that the apex is not visualized in the roentgenogram. The nonvisualization of the petrous apex does not indicate a perforation. Perforation of the apex, however, may occur.

2. Subacute cases of coalescent petrositis, with adequate drainage, not requiring immediate operation for drainage of the petrous apex. The same roentgen changes are shown as in acute coalescent petrositis with inadequate drainage (fig. 4). The typical symptom complex described by Kopetzky and Almour is absent; otorrhea is present. These cases have a tendency to become chronic.

3. Chronic cases, presenting persistent otorrhea due to an infection in the petrous apex and showing roentgen evidences of chronic osteitis. There may or may not be complete obliteration of all air cells (fig. 5).

Follow-up studies of several of the operatively cured patients showed productive changes in the apical portions of the petrous pyramids with complete obliteration of the air cells. There was no visualization of pneumatic structure in the apex. There was no restitution to normal. The petrous apex appeared dense and sclerotic.

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2. These findings refer to the roentgen appearance of the petrous apex as observed in a film of the basis cranii. (Additional studies were made with oblique positions of the head in some of the cases.)

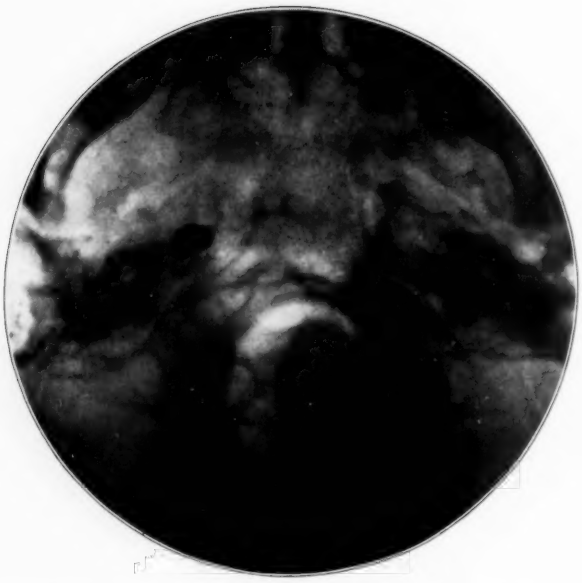

Fig. 5.—Persistent otorrhea following mastoidectomy. There are productive changes in the petrous pyramid. The apex shows a diminution in aeration and thickened trabeculae. Iodized poppy seed oil 40 per cent injected into the mastoid antrum during the revision operation outlined the tract of infection which extended into the petrous apex.

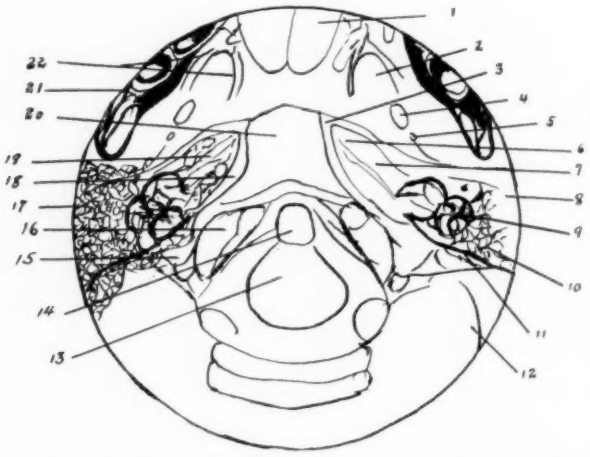

Fig. 6.—*1*, sphenoid sinus; *2*, pterygoid fossa; *3*, middle lacerated foramen; *4*, foramen ovale; *5*, foramen spinosum; *6*, petrous apex (cells destroyed); *7*, carotid canal area; *8*, area of resected mastoid; *9*, labyrinthine area; *10*, cells in basal portion of pyramid; *11*, superior petrosal sinus; *12*, lateral sinus; *13*, foramen magnum; *14*, odontoid process of axis; *15*, posterior lacerated or jugular foramen; *16*, occipital condyle; *17*, mastoid cells; *18*, inferior petrosal sinus; *19*, petrous apex (pneumatized); *20*, basilar process of occipital bone; *21*, mandible; *22*, pterygoid plates.

The twenty cases of suppuration of the pneumatic petrous pyramid were grouped as follows:

1. Acute cases. Sixteen cases presented the Kopetzky-Almour syndrome, with the characteristic roentgen findings of acute coalescent petrositis.

2. Subacute cases. Two cases showed roentgen evidences of coalescent petrositis and no clinical symptoms other than otorrhea.

3. Chronic cases. Two cases showed evidences of suppuration in the petrous apex of a chronic nature. (Investigation of chronic otorrhea would probably reveal many more such cases.)

It is important to note that the subdivisions acute and subacute are based on clinical symptoms and not on roentgen findings. It is advisable to have the clinical diagnosis of empyema of the petrous apex supplemented by roentgen evidence of empyema of the petrous apex.

SUMMARY

Roentgenographically the pneumatic petrous pyramid shows variations from the normal when an otitic infection is present. These variations do not always indicate a suppurative lesion of the petrous apex.

The roentgen appearance of acute coalescent petrositis or empyema of the petrous apex is that of diminished aeration, halisteresis of the apex, loss of trabeculations and sometimes solution of continuity.

There is no roentgenologic distinction between the acute and subacute types of coalescent petrositis; the differentiation is clinical.

Chronic petrositis shows productive changes in the apex.

The cases of healed acute coalescent petrositis show productive changes with complete obliteration of air cells.

333 West End Avenue.

HEMORRHAGE FROM PHARYNGEAL AND PERITONSILLAR ABSCESSES

REPORT OF CASES, RÉSUMÉ OF THE LITERATURE AND DISCUSSION OF LIGATION OF THE CAROTID ARTERY

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When one for the first time encounters a severe, perhaps fatal, hemorrhage in connection with an abscess in the throat the first reaction is that of surprise or shock, as the occurrence is unforeseen and wholly unexpected. To one who has successfully treated a large number of patients with retropharyngeal and peritonsillar abscesses over a period of years without untoward results, it is a matter of deep concern to be faced with an alarming hemorrhage or a fatality. As a result one turns to the literature for information on the subject, as textbooks in the main make very little reference to this grave complication, and one is rather surprised to discover that the apparently rare complication is really not so infrequent and that the topic has been freely written about, discussed and investigated many times. No doubt every member of this society has at some time or other encountered such cases, and were it possible to collect and analyze all of these experiences in detail they would undoubtedly afford most valuable data for future guidance.

Chronologically, the most significant articles on this subject have been contributed by the following investigators: Lebram (1906), Newcomb (1908), Lubbers (1912), Stumpf (1920), Sercer (1928), Lifschutz (1931), Skoog (1932), Le Mesurier (1931), Gerlings (1932) and Brauer (1933). In addition, there have been numerous case reports with appropriate comment. Yet, despite the fact that these essays have contributed most valuable information, we have felt, in view of our personal experiences, that a more comprehensive review of the subject which would cover all phases might lead to a clearer understanding of it and help point the way to more definite conclusions. We believe that it is not only important to know where the infection is located and how it got there, but even more so to know approximately where the hemorrhage originated and how dangerous it may be. We are persuaded that the question goes beyond peritonsillar or retropharyngeal abscesses

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alone and embraces infection of any sort beginning in the throat in which secondary pathologic changes in adjacent structures lead to hemorrhage whether from the ear, the throat or the neck. This is our reason for including hemorrhages from the internal jugular vein in cases of scarlet fever.

On analyzing the various groups, we have found that there are certain fairly constant factors in the history, the progress of the case and the local manifestations which point the way to a correct diagnosis of the source of the hemorrhage, and that when these facts are taken into consideration in connection with a knowledge of the anatomy, one may be permitted certain fairly well defined deductions as to treatment and prognosis. It is important to be able to differentiate a minor hemorrhage due to involvement of a branch of the ascending pharyngeal or the tonsillar artery arising in the course of a peritonsillar abscess from a hemorrhage due to erosion of the internal carotid or the superior thyroid artery. In the one instance unnecessary ligation of a major vessel may be avoided, whereas in the second instance delay in such radical treatment may lead to a fatality.

It is our purpose, therefore, to present first the cases which came under our direct supervision, including a number from the records of the Michael Reese Hospital, as well as personal communications of interested colleagues; secondly, we shall discuss the anatomic and pathologic features of the various types of abscesses that may be encountered, including the factors involved in a complicating hemorrhage; thirdly, we shall present a résumé of 231 cases published in the literature (including those described) analyzing them by groups according to pathologic changes, autopsy reports and results of therapy; fourthly, we shall attempt to set forth our conception of the diagnosis of the probable source of the hemorrhage according to clinical findings and outline our idea of the therapeutic procedure in each instance, and, lastly, we shall consider ligation of the common carotid artery in general, as well as in relationship to the therapy of certain groups.

AUTHORS' CASES

Case 1 (Salinger).—A boy, aged 3½, had a sore throat for one week and spontaneous evacuation of pus from the throat with slight recurring hemorrhage for four days. Then two severe hemorrhages occurred within eight hours, amounting to about 8 and 12 ounces (236 and 355 cc.) each. There was a small opening in the retropharyngeal swelling on one side from which a blood clot protruded. There was no definite external swelling. The child was very pale and listless; the pulse rate was 150 and thready.

The common carotid artery was ligated with the patient under local anesthesia, following which he was given a transfusion of 300 cc. of whole blood. After operation the patient had a slightly irregular temperature. Horner's syndrome and hypoglossal paresis ensued. The former subsided within a few weeks,

but the latter was permanent, resulting in hemiatrophy of the tongue. Recovery was complete. The tonsils and adenoids were removed three months later without untoward complications.

CASE 2 (service of Jerome F. Strauss).—A girl, aged 5, was ill for three weeks with a sore throat and enlargement of the cervical glands. An abscess (cervical) was incised at the end of fourteen days. There was temporary improvement. The swelling then increased, and bleeding began one week later. A retropharyngeal abscess was discovered. There was bleeding off and on for six hours. Preparations were made for ligation. A violent hemorrhage occurred while the patient was being taken to the operating room, and she died. Autopsy was not performed.

CASE 3 (service of Jerome F. Strauss).—A boy, aged 7, had a sore throat for eleven days. Spontaneous bleeding from the nose and throat occurred on the ninth day. He had fever and chills. There was a retropharyngeal abscess with parapharyngeal involvement. Bleeding was intermittent for several days. He was taken to the hospital, and the common carotid artery was ligated. Severe hemorrhage occurred the next day. Ligation of the internal jugular vein was performed, and the bulb was packed from above. The patient died.

Autopsy showed diffuse cellulitis of the neck, cervical adenitis and a marked fibrous exudate opening in the pharynx behind the tonsil. Clots were removed; this exposed a cavity the lining of which was continuous with that of the common carotid artery.

CASE 4 (service of A. Levinson).—A girl, aged 2, had a retropharyngeal swelling. Spontaneous rupture of the abscess occurred while the patient was on the way to the hospital. The opening in the abscess was enlarged, and there was a bloody discharge. The glands were enlarged. There was dyspnea. Tracheotomy was performed the next day. There was continuous bleeding from the throat. Ligation of the superior thyroid artery was performed, and the bleeding diminished. The patient's general condition was poor, and she died.

Autopsy revealed cellulitis and fibrinous exudate in the tissues of the neck, mostly in the parapharyngeal space and the pyriform sinus. The condition of the vessels was not noted.

CASE 5 (service of Harry Kahn).—A boy, aged 10, had a sore throat for one week, a retropharyngeal abscess on the right and submaxillary swelling. The abscess was incised, but no pus was obtained. Two days later hemorrhage occurred from the throat. This recurred the next day. The patient died. Autopsy was not performed.

CASE 6 (Pearlman).—A boy, aged 4, had a swelling of the left side of the neck and a retropharyngeal abscess for seven days. There was a rise in temperature. The abscess was incised, but no pus was obtained. Forty-eight hours later a sudden foudroyant hemorrhage occurred, but death took place in a few minutes. Autopsy was not performed.

CASE 7 (Pearlman).—A girl, aged 5, was ill for one week. There was swelling of the left side of the throat and neck. A spontaneous hemorrhage occurred from the throat, a large amount of blood being lost. Ligation of the common carotid artery was performed. Hemorrhage recurred the next day. The wound reopened. The carotid artery was again ligated. A perforation in the pharynx was found leading into a large ulcerated cavity. The cavity was packed, and there was no more bleeding. Myosis appeared after the first ligation, but disappeared later. There was slight mental confusion for a few hours. Recovery was complete.

Case 8 (Pearlman).—A boy, aged 6, had a retropharyngeal and parapharyngeal abscess. He was ill for seven days. An incision was made in the throat, but no pus was obtained. There was moderate bleeding for one hour. Hemorrhage recurred the next day in large amounts. Ligation of the common carotid artery was performed, with complete recovery.

Case 9 (Guttman).—A boy, aged 5½, had a retropharyngeal abscess. The neck was swollen. There were several spontaneous hemorrhages from the throat. Contralateral facial paresis and hemiplegia came on spontaneously. Two days later ligation of the common carotid artery was performed. The hemorrhage ceased, and the paresis disappeared within a few days. Recovery was complete.

Case 10 (Lorance to Salinger).—A girl, aged 4, had a peritonsillar abscess, which was incised. Ten days later there was bleeding from the throat. An explosive hemorrhage followed. A transfusion of blood was given, but the patient died. Autopsy was not performed.

ANATOMY AND PATHOLOGY

Pharyngitis or tonsillitis, whether occurring as a part of a nonspecific infection of the upper respiratory tract or as one of the contagious diseases, may lead to suppuration in the adjacent tissues; as a result, the familiar retropharyngeal and peritonsillar abscesses occur.

In retropharyngeal abscess the infection is carried by lymphatics to the small lymph glands lying anterolateral to the upper cervical vertebra, which break down, causing an accumulation of pus which, as a rule, is circumscribed within the pharyngeal limits, but which in some instances may follow the deep fascial planes up to the base of the skull or down to the mediastinum.

In peritonsillar abscesses, as is well known, the infection is carried beyond the capsule of the tonsil and is usually limited to the immediate vicinity by the fascia of the middle constrictor muscle.

Infection of the parapharyngeal spaces, which has lately been the subject of widespread study, is in most instances blood-borne through thrombosed veins leading from the tonsillar plexus to the tissues external to the middle constrictor muscle and from there may spread to the submaxillary gland, the carotid sheath and the parotid spaces.

For some time there has been a difference of opinion as to whether the thrombophlebitis is the etiologic factor or whether, as some authors insist, the infection is carried by the lymphatics to the glands adjacent to the large vessels of the neck. There has been ample anatomic, pathologic and histologic proof submitted by Fränkel, Uffenorde, Zange, Claus, Waldapfel and others to support both theories, so that one must conclude that the pathways of infection are both lymphatic and venous, as well as by direct contiguity of tissue, depending on the type of infection.

One therefore has to consider infection in several different areas adjoining the pharyngeal mucosa in any one of which certain factors

may operate in the production of a single hemorrhage or a series of hemorrhages that may prove fatal. In studying the factors operating toward this end, we shall consider first the anatomy of the area involved and, secondly, the type of infection and its mode of progression.

We shall deal only briefly with the first of these topics because we believe that it is generally understood by experienced laryngologists. The classic work of Mosher in 1929 and the more recent elaboration of A. L. Beck demonstrating in detail the fascial compartments in the neck and their relationship to each other as well as to the pharyngeal mucosa are too well known to require more than appreciative mention.

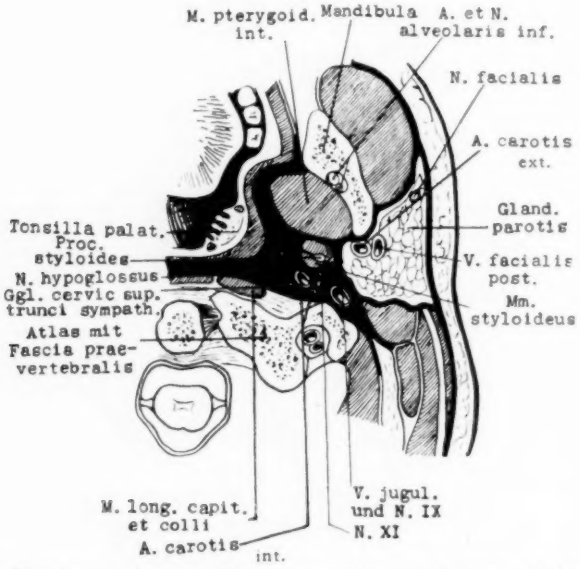

Fig. 1.—Parotid recess and topography of parapharyngeal space. (After Corning.)

We desire in addition merely to call attention to a drawing reproduced from Corning (fig. 1) which shows graphically the relationship of the vital structures in a cross-section of the neck at the level of the tonsil. The picture shows how one layer of the deep fascia follows the submucosa of the pharynx, separating it from the constrictors and deeper muscles externally, and further how another layer splits at the styloid process to separate the parapharyngeal space into its anterior and posterior compartments. Still further, the picture shows how the deep vessels of the neck pass through the posterior compartment and how much closer to the pharyngeal wall the internal carotid artery lies than does the external carotid artery, which is partly shielded by the styloid process and the muscles descending from it. One need only recall in addition that the external carotid artery, giving off its branches, becomes

smaller as it ascends through the parapharyngeal space, while the internal carotid artery, giving off no branches in this region, loses none of its caliber as it approaches the base of the skull. As to the lymphatics, a brief study of a plate from Most's excellent work on this subject (fig. 2) discloses definitely the numerous lymph channels leading to the

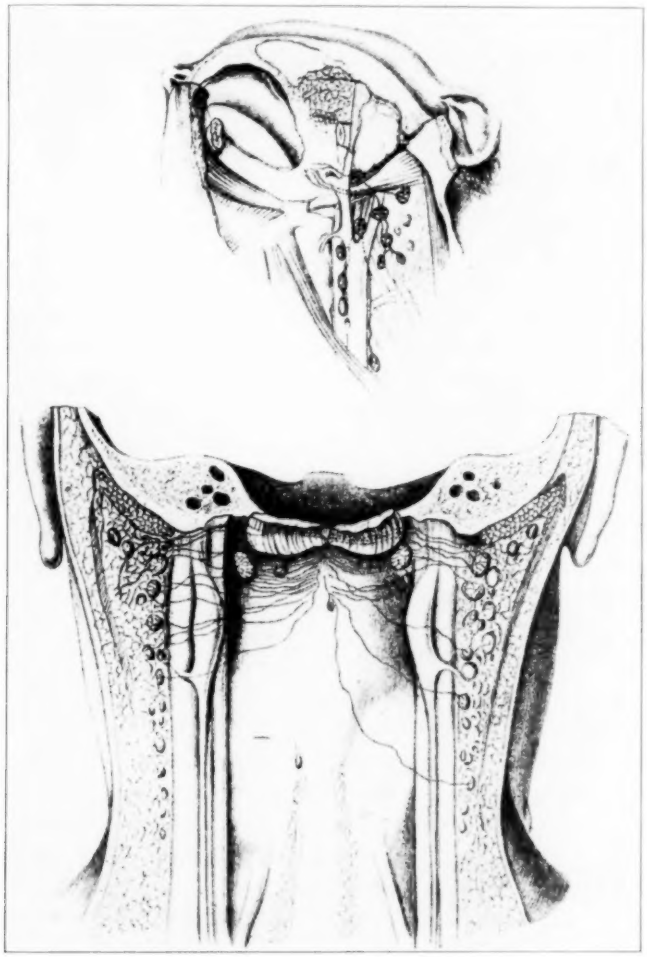

Fig. 2.—Lymphatics and glands of the pharynx. (After Most.)

retropharyngeal glands and from there to the deep cervical glands, as well as the other channels draining the tonsillar area which reach the same ultimate depot. These are simple well known facts. The following facts are less well known, although they are frequently enough alluded to in the literature: (1) The internal carotid artery normally makes several curves in its course in the neck which may become exag-

gerated into tortuosities that will bring it into close proximity with the pharyngeal mucosa, and (2) it is frequently the site of an aneurysmal dilatation.

Jastram called attention to the fact that the internal carotid artery makes five distinct curves during its course which, taken together with the absence of branches in its cervical portion, predisposes it to dilatation and tortuosities. He listed the normal curves as follows: (1) a slight S-shaped curve just behind the bifurcation, (2) a horizontal curve with its convexity forward and medial just before its entrance into the skull, (3) the curve within the carotid canal, (4) a curve in the sulcus caroticus near the sella and (5) a slightly S-shaped curve, a convexity anterior in the impressio carotica of the sphenoid.

Schaeffer, in 1921, commenting on a letter in *The Journal of the American Medical Association* reporting fatal hemorrhage from an aberrant internal carotid artery during tonsillectomy, said that he knew of 3 similar cases. He submitted a photograph of a postmortem specimen showing a tortuous internal carotid artery in close proximity to the tonsil. Further, he cited 22 additional references to similar cases in the literature. In his summary he makes the following points:

1. Palpation will disclose the vessel when it is close to the pharyngeal wall.
2. Visible pulsation is usually caused by the internal carotid artery, but may also be due to the ascending pharyngeal or even the external carotid artery.
3. The redundant or tortuous internal carotid artery and agenesis of the internal carotid artery represent reversion to the primitive type.
4. Age has no bearing on the development and presence of a tortuous internal carotid artery.
5. Tortuosity is more common in the female.

Since the publication of Schaeffer's article there have been numerous additional references to this anomaly in the literature. Poppi reported a case of complete absence of the internal carotid artery. Streit noted pulsation in the pharynx of a man, aged 66, who died of apoplexy. He obtained permission for autopsy and found dilatation of both internal carotid arteries from the bifurcation upward, a loop bringing the arteries in close proximity to the posterior pillar of the tonsils (fig. 3).

In 1929 Carmack reported 5 cases of fatal fulminating hemorrhage during or after tonsillectomy; he assumed from the severity of the bleeding that they must have been due to anomalously placed internal carotid arteries. He showed an anatomic specimen in which the internal carotid artery extended within 3 mm. of the posterior pillar and another in which it was 1 cm. distant, the normal distance being from 2 to 2.5 cm. Further, he reported having seen 2 cases of pulsating arteries on the posterolateral wall of the pharynx which he felt certain were the internal carotid arteries. The article is complete and includes 42 references to the subject.

Aneurysm of the internal carotid artery has been repeatedly noted in the literature, in most cases as being traumatic, although there are several instances in which a previously undiagnosed aneurysm was mistaken for a retropharyngeal abscess which on incision resulted in fatal hemorrhage. These true aneurysms must not be confused with the false aneurysm that has been observed far more frequently in connection with the infection of the throat and neck; it will be more thoroughly discussed in a later paragraph.

True aneurysm of the carotid arteries is comparatively rare. Crisp found only 25 involving the carotid arteries in a total of 551 aneurysms. Wyeth, in a series of 789 ligations of the common carotid artery found

Fig. 3.—Dilated internal carotid arteries due to constricting loops. (After Streit.)

only 4 cases of aneurysm of the internal carotid artery. Barwell said that 87.2 per cent of all carotid aneurysms affect the common carotid artery, 7 per cent the external carotid artery, and 5.7 per cent the internal carotid artery. Monod and Vanverts cited 8 cases and Bobbio 18 cases. Other cases of aneurysm have been described by Agnew, Blauel, Booth, Briggs, Callander, Crisp, del Fabro, Franz, Kümmel, Lichatschoff, Mendes and Werner.

Winslow collected 70 cases of aneurysm of the carotid arteries and found that 36 were spontaneous, 13 traumatic and 10 arteriovenous, and that 10 were caused by erosion. Most of the aneurysms were recognized and a diagnosis was made, but in a few cases, unfortunately, the diagnosis was not made until an intercurrent infection of the throat had brought the patients under observation. Frequently the presence of a soft swelling in the throat or neck led to the mistaken diagnosis of an

abscess with fatal results from incision. As a rule, aneurysm of the internal carotid artery presents in the lateral part of the pharynx behind the tonsil, whereas aneurysm of the external carotid artery is found in the neck. In the presence of infection one must always view with suspicion any soft, smooth, fluctuant swelling presenting in the lateral part of the pharynx or at an angle of the jaw where pulsation is present or where there is a difference in the pulse of the two sides. A preliminary needle puncture may be a most valuable means of diagnosis, although at times a negative puncture does not exclude aneurysm because of the possibility of the sac being lined with heavy coaguli.

False aneurysm, particularly of the internal carotid artery, has been encountered much more frequently as a result of septic destruction of the arterial wall and constitutes one of the most striking pathologic entities in the series of cases of hemorrhage under consideration. In 24 of the 90 autopsies in our series the presence of a false aneurysm was noted.

The genesis, development and diagnosis of this extremely dangerous complication will be more thoroughly discussed in a later paragraph. Suffice it for the moment to point out the grave significance of an infection occurring in the presence of such complications as an aberrant internal carotid artery or an aneurysm, factors which, if unrecognized, may easily lead to a fatality unless sufficiently radical and prompt measures are instituted. False aneurysm is a pathologic entity which may be present in any abscess of the throat that bleeds; knowledge of this fact will always put one on guard against underestimating the gravity of the situation.

ANALYSIS OF 227 CASES

In attempting to analyze the series of 227 cases which we have collected we felt that we could obtain the most information by segregating them into groups according to the anatomic spaces involved, as each group presents a somewhat different pathologic anatomy from the others, and the results of surgical intervention or nonintervention vary considerably among them (table 1).

Group 1: Peritonsillar Abscess.—There were 85 cases in this group in the course of which there were more or less severe hemorrhages: Twenty-eight patients were subjected to ligation of either the common or the external carotid artery. It may be assumed from the records that in general an intervention of this sort was undertaken for bleeding that must have been severe. Of the 11 patients on whom ligation of the external carotid artery was performed, 3 died (37.5 per cent); of the 17 on whom ligation of the common carotid artery was performed, 2 died (13.3 per cent). Fifty-eight patients bled and received no surgical therapy apart from local measures such as pressure, ice compresses and enucleation of the tonsil; 29 (50 per cent) of these

patients died. Aside from a few cases reported by Brauer, in which the bleeding was not especially severe, we may safely assume that in the remainder the hemorrhage was more or less alarming, else it is not likely that the incidents would have been reported. Although the total number of cases is too small to serve as a basis for actuarial statistics, nevertheless they do show that the surgeons availing themselves of ligation, especially of the common carotid artery, seem to have had the best results. If one believes that some of the recoveries following ligation can be explained by assuming involvement of the smaller vessels and that recovery would possibly have taken place anyway, as indicated by the 29 recoveries in cases in which ligation was not performed, one must not forget that there were an equal number of deaths (29) in the latter group. Certainly this last fact proves that the surgeon under-

TABLE 1.—*Results of Ligation by Types of Abscess*

		Ligation of External Carotid Artery		Ligation of Common Carotid Artery		No Ligation	
Abscess	Number of Cases	No. of Patients Who Recovered	No. of Patients Who Died	No. of Patients Who Recovered	No. of Patients Who Died	No. of Patients Who Recovered	No. of Patients Who Died
Peritonsillar	85	8	3	15	2	29	29
Peritonsillar and parapharyngeal	31	3	1	5	1	4	17
Retropharyngeal	32	0	2	2	3	1	24
Retropharyngeal and parapharyngeal	28	0	1	8	6	0	13
Parapharyngeal	21	1	0	3	4	1	12
Cervical	30	0	0	2	3	1	24
Total	227	12	7	35	19	36	119

estimated the severity of the hemorrhage and the size of the vessel from which it sprang.

Group 2: Peritonsillar Abscess Complicated by Involvement of the Parapharyngeal Space.—There were 31 cases in this group, in 10 of which ligation was performed. Four ligations of the external carotid artery and 6 of the common carotid artery were performed with 1 death from each. Seventeen of the 21 patients on whom no ligation was performed died. From this it is seen how much more grave is the prognosis in the cases in which ligation is not performed, a fact that can be ascribed only to a more general involvement of the major vessels in the suppurative process by reason of its extension to the parapharyngeal space.

Group 3: Retropharyngeal Abscess.—The figures in this group are even more significant. Ligation was not performed on 25 of the 32 patients who bled, and all but 1 died. There were ligations of the external carotid artery in 2 cases which ended fatally. In 5 cases the common carotid artery was ligated, with recovery in 3.

Group 4: Retropharyngeal Abscess Complicated by Involvement of the Parapharyngeal Space.—There were 28 cases in this group. In 13 cases there was no ligation, and all of the patients died. In 14 cases the common carotid artery was tied, and 8 patients recovered. There was ligation of the external carotid artery in 1 case which ended fatally.

Group 5: Parapharyngeal Abscess (Cases in which the histories do not indicate the presence of either a peritonsillar or a retropharyngeal abscess).—Thirteen of 21 patients had no ligation performed, and all but 1 died. In 7 the common carotid artery was ligated, and 4 of these patients died. In the case in which ligation of the external carotid artery was performed the patient recovered.

Group 6: Cervical Abscess (or Cellulitis).—There were 30 patients in this group suffering mostly from postscarlatinal cervical cellulitis with suppuration and gangrene. In 5 the common carotid artery was ligated, with 2 recoveries. The remainder of the patients died. Surgical exposure and autopsies in most of these disclosed the source of the hemorrhage as the internal jugular vein.

Of a total of 227 cases for all groups, there were 154 in which no ligation was performed, and only 36 patients (23 per cent) recovered. Ligation was performed on 72 patients, and 47 recovered (65 per cent). There were 18 ligations of the external carotid artery, with 11 recoveries (61 per cent), and 54 ligations of the common carotid artery, with 36 recoveries (67.5 per cent).

These figures prove definitely that the advantage was in favor of the patients on whom a ligation was performed. The high mortality in the cases in which ligation was not performed was shown in 90 autopsies to have been due to erosion of a major vessel (the internal carotid artery in most cases), which had been involved in the suppurative process with resultant weakening of its walls and aneurysmal dilatation.

The postmortem observations may be studied first with a view toward ascertaining which vessels the individual inflammations of the throat and neck attack by preference (table 2).

Group 1.—One notices that in 15 autopsies following death from peritonsillar abscess the internal carotid artery was involved 11 times (4 of these were associated with false aneurysm), and in 4 instances other vessels were eroded. By other vessels we mean branches of the external carotid artery, such as the superior thyroid, the superior laryngeal and the ascending pharyngeal. The common carotid and the external carotid arteries were not invaded once in this group.

Group 2.—When peritonsillar abscesses were associated with a spread to the parapharyngeal space, the internal carotid artery was found eroded 7 times, twice with the formation of a false aneurysm,

and the external carotid artery, the common carotid artery and the other vessels each 3 times.

Group 3.—Simple retropharyngeal abscess attacked the internal carotid artery by preference; 14 times in our series, 3 being associated with a false aneurysm. The common carotid artery was involved twice, and the other arteries 5 times.

Group 4.—Autopsies in cases in which death resulted from retropharyngeal abscess associated with involvement of the parapharyngeal space showed the internal carotid artery eroded 8 times, in 7 of which there was also a false aneurysm. Two other cases revealed erosion of the common carotid artery.

TABLE 2.—*Autopsy Observations*

Abscess	Erosion of Internal Carotid Artery	Erosion of External Carotid Artery	Erosion of Other Arteries	Erosion of Common Carotid Artery	Erosion of Internal Jugular Vein	False Aneurysm and Erosion of Internal Carotid Artery
Peritonsillar	7	0	4	0	0	4
Peritonsillar plus parapharyngeal	5	3	3	3	0	2
Retropharyngeal	11	0	5	2	0	3
Retropharyngeal plus parapharyngeal	1	0	0	2	0	7
Parapharyngeal	5	1	2	1	1	3
Cervical	1	0	0	1	13	0
Totals	30	4*	14†	9‡	14§	

* One false aneurysm of the external carotid artery was included among the erosions.
† One false aneurysm of the internal maxillary artery was included with erosion of the other vessels.
‡ Three false aneurysms of the common carotid artery were included among the erosions.
§ There was one erosion of the transverse sinus (not included with erosion of the jugular vein).

Group 5.—Autopsy in cases of parapharyngeal abscess in which death was caused by hemorrhage revealed erosion of the internal carotid artery in 8 cases, in 3 of which it was associated with a false aneurysm. In the remaining 5 cases, the external carotid artery was eroded once, other arteries twice, the common carotid artery once and, what is very unusual, the internal jugular vein once.

Group 6.—The cervical infections accompanying such illnesses as scarlatina and producing large sloughs in the neck attacked by preference the internal jugular vein 13 times in a total of 15 cases. Of the other 2 cases, 1 showed erosion of the internal carotid artery and 1 erosion of the common carotid artery.

In 90 postmortem examinations, the vessel found to be most frequently involved was the internal carotid artery—49 times; in 19 cases the involvement was associated with a false aneurysm. The external carotid artery was eroded in 4 cases; in 1 of these a false aneurysm was present. Other vessels were attacked in 14 cases, and in 1 of these,

in which the internal maxillary artery was concerned, a false aneurysm was also present. The common carotid artery was eroded in 9 cases; in 3 the erosion was accompanied by a false aneurysm. One examination revealed erosion of the transverse sinus, a most unusual finding.

With the omission of the venous hemorrhages, which in a sense belong in a class by themselves, there were in this series 76 deaths due to erosion of the arteries. If, with an eye toward therapeutic intervention, one groups together the cases of erosion of the internal carotid and common carotid arteries, they number 58, by far the largest majority, leaving only 18 in which benefit could be had by ligation of the external carotid artery or of one of its branches.

In the light of these facts it becomes less difficult to understand the significance of the hemorrhages. By referring to the group analysis, it is seen how much gravity a case assumes once the infection passes the usual confines of a peritonsillar abscess and invades the parapharyngeal spaces. Even the simple cases of retropharyngeal abscess in which involvement of the parapharyngeal spaces was definitely not noted proved overwhelmingly fatal once a hemorrhage ensued and prompt surgical intervention in the shape of ligation was deferred. The reason for this lies in the fact, already mentioned, that the internal carotid artery is in close proximity to the area involved, and also because a limiting membrane or fascial plane separating the retropharyngeal from the posterior compartment of the parapharyngeal space has not been definitely proved.

As for the incidence of single or multiple hemorrhages, the figures also furnish light on the problem of surgical intervention. In 85 cases there was only one hemorrhage, and in some instances it was of such magnitude as to prove immediately fatal. Undoubtedly some of the patients could have been saved had the presence of a false aneurysm been diagnosed. In several instances in which the surgeon suspected aneurysm and exploratory puncture confirmed the suspicion, immediate ligation saved the patient. Spontaneous recovery occurred in only 8 of the 85 cases, and in several of these the hemorrhage was not particularly severe, arising as it did from the smaller branches of the external carotid artery. Fourteen of the 24 patients on whom ligation was performed after the first hemorrhage were saved.

In the consideration of the cases in which there were several hemorrhages occurring at intervals, we found it more difficult to analyze the situation because of the varying pathologic conditions present and because the amounts of the successive hemorrhages could not be accurately estimated from a mere reading of the reports. It should be sufficient, however, for one to realize that recurrent hemorrhages of fair magnitude in the presence of definite swelling of the neck, when a peritonsillar or

a retropharyngeal abscess has been diagnosed, indicate involvement of a major vessel and that no time should be lost in performing ligation before a final and fatal hemorrhage ensues.

The occurrence of spontaneous hemorrhages from the ear is of particular importance. We found it noted in 20 cases, in 16 of which the outcome was fatal. The accompanying autopsy reports, when available, showed in every instance that there was a false aneurysm of the internal carotid artery with erosion either into the eustachian tube or into the external auditory canal by way of the parotid space. Consequently when this symptom is present one can safely assume the presence of an aneurysm and proceed at once to ligation before the sac ruptures into the throat.

Brauer has attempted to define the source of the hemorrhages from their character. He divided them into six groups.

1. Hemorrhage from the ear; false aneurysm of the internal carotid artery.

2. Sudden single foudroyant hemorrhage; false aneurysm of the internal carotid artery.

3. Several minor prodromal hemorrhages followed by a foudroyant fatal one; false aneurysm of the internal carotid artery. This group is differentiated from group 5 by persistent, even increasing, dense swelling in the throat or the neck.

4. Several severe hemorrhages in succession, generally from branches of the external carotid artery.

5. Recurrent slight to moderate hemorrhages due to local vessels of the tonsillar bed or the palate.

6. Venous hemorrhage.

We agree with Brauer in his classification with the exception of his interpretation of group 4. Recurrent hemorrhages, whether slight or severe, may just as well be due to a false aneurysm as to involvement of the minor vessels or even branches of the external carotid artery, as was revealed in a number of cases in our series. One must go further than a mere notation of the number and severity of the hemorrhages and take into consideration the accompanying physical findings. Just as in group 3 he points out the significance of increasing dense swelling in the throat or the neck, a swelling that increases as soon as the hemorrhage stops, so in group 4 the presence of evidence of parapharyngeal cellulitis may indicate that involvement of either the external or the internal carotid artery is just as probable as involvement of one of the branches of the external carotid artery.

Finally, when successive hemorrhages occur from a retropharyngeal abscess, whether they are moderate or severe, one cannot dismiss the probability of involvement of the internal carotid artery as the most likely source of the bleeding.

THERAPY

Having thus shown the connection between certain types of pathologic changes and resultant hemorrhages and having furthermore proved the urgent necessity for prompt radical treatment, we shall next consider ligation. The following questions arise: Which vessel should be ligated, and what are the dangers arising from this procedure? The answer to the first question lies in the pathologic type involved. One must first exclude the cases of Brauer's group 5, in which minor hemorrhages occur from the tonsillar bed owing to the erosion of the smaller vessels. This group should include only minor hemorrhages following incision of the abscess, in which the hemorrhage can be seen to be issuing from the margins of the incision, from a crypt or from the margin of the palate itself. Only in this type of case is it safe to rely on palliative measures such as ice compresses, packing, pressure, enucleation of the tonsils and ligation of the bleeding vessels in the tonsillar bed.

As has been pointed out by Skoog and by Sercer, the dangerous cases are those in which one or more of the following factors are present:

1. Spontaneous hemorrhage or hemorrhages so severe as obviously not to be arising from a minor vessel.

2. A protracted course in which the swelling fails to disappear following a previous incision.

3. Hematoma of the surroundings as evidenced by submucous discoloration or tense, brawny swelling.

4. Increasing pain, swelling locally and in the neck and trismus, despite incision of the abscess and long after the course of a normal peritonsillar abscess has been run.

5. The presence of pulsation in the peritonsillar area.

If any of these factors is definitely established, we recommend exposure of the carotid sheath and, if the condition of the patient is not too critical, a search for the source of the hemorrhage. In the minority of cases one of the branches or the stem of the external carotid artery will be at fault. In this event the external carotid artery or such a branch should be ligated. If one fails to locate such a pathologic change, the only safe procedure is ligation of the common carotid artery.

The figures previously presented prove that in the majority of cases the lesion is in the internal carotid artery. Another alternative presents itself. If the patient's general condition is such that the risk of another hemorrhage is not too great, one may ligate the external carotid artery and place a loop about the common carotid artery, leaving the wound wide open so that the latter may be promptly tied in the event of a recurrence of the hemorrhage. A competent attendant would have to

be present night and day at the patient's side, not leaving him for one moment until all danger was past.

Coming to the discussion of procedure in the case of retropharyngeal abscess, alone or complicated by changes in the parapharyngeal space, we see no other alternative than ligation of the common carotid artery. A study of the case histories herewith presented and the postmortem reports demonstrates unequivocably the rationale of such a stand.

As to infections of the parapharyngeal space without a concomitant retropharyngeal or peritonsillar abscess, we are again confronted with evidence showing the preponderance of involvement of the internal carotid artery, and here, also, we should feel impelled to undertake the more radical procedure of ligating the common carotid artery.

In regard to cervical infections, in which it has been shown that the vast majority of hemorrhages are due to erosion of the internal jugular vein, the only therapy that can possibly be of avail is wide exposure of the cervical spaces and ligation of the internal jugular vein, if accessible; otherwise, one has to rely on tight packing for the control of the hemorrhage. Rarely has there been erosion of one of the carotid arteries in this type of infection, and therefore one is not justified in ligating either one of the arteries unless there is definite evidence of a false aneurysm or of erosion. The outlook in this type of case is uniformly bad because of the severe accompanying toxemia and local edema which frequently necessitates tracheotomy.

Having recommended ligation of the common carotid artery in such a wide range of cases, we have to consider the risks attending such therapy. With this in view we have undertaken a study of the literature on this subject.

LIGATION OF THE COMMON CAROTID ARTERY

When confronted with the necessity for ligating the common carotid artery, one is necessarily impressed with the seriousness of the procedure in view of the fact that it entails a high mortality and therefore is not to be undertaken too lightly. A survey of the literature on this subject reveals an array of imposing articles, most of them dating back to the middle of the last century, with the contributions becoming less and less frequent and of narrower scope as one approaches the present. We have studied all of these publications in detail and have found that necessarily there has been considerable overlapping in the successive series of cases reviewed, so that one can draw deductions from a mere summary of their totals. Nevertheless, we have collected them in tabular form (table 7) because they represent the mature experience of almost a century, and offer a basis of comparison with the conclusions we have been able to draw from the series of cases of

TABLE 3.—*Causes of Fatalities from Ligation of the Common Carotid Artery*

	Sepsis	Secondary Hemorrhage	Severe Constitutional Disturbances	Intercurrent Diseases	Intracranial Complications	Other Causes
Siegrist..... 371 deaths	74 cases 20%	62 cases 17%	42 cases 11%	15 cases 4%	80 cases 22%	Insufficient data, 98 cases; 26%
Le Fort..... 17 deaths; aneurysm only	1 case 5.8%	6 cases 35%	1 case 5.8%	.....	7 cases 40%	Pressure on trachea, 2 cases; 12%
Wyeth..... 200 deaths	.....	44 cases 22%	23 cases 11%	.....	69 cases 34%	22 cases of bleeding plus other causes; 11%

TABLE 4.—*Results of Ligation of the Common Carotid Artery by Decades (Authors' Series)*

Age	Number of Cases	Number of Patients Who Recovered	Number of Patients Who Died	Number of Cranial Complications
Up to 10.....	19	11	8	4
10 to 20.....	8	4	4	1
20 to 30.....	12	9	3	3
30 to 40.....	5	2	3	2
Over 40.....	4	2	2	1
Age not stated.....	6	5	1	0
	54	33	21	11

TABLE 5.—*Percentage of Fatalities from Ligation of the Common Carotid Artery by Ages*

	Up to 10	11 to 20	21 to 30	31 to 40	41 to 50	51 to 60	Over 60	Comment
Pilz.....	18	23	32	32	50	50	79	All cases
Siegrist.....	14	35	38	37	52	75	92	Cases of hemorrhage only

	Up to Age 20	21 to 40	Over 40	Age Not Given
Salinger and Pearlman	37	40	50	18

TABLE 6.—*Analysis of Fatalities from Ligation of the Common Carotid Artery by Cranial Symptoms*

Pilz, 320 cases	Hemiplegia	Monoplegia or Diplegia	Convulsion	Headache; Vertigo; Transient Loss of Consciousness	Sensory Disturbance	Coma	Delirium	Other Symptoms
Cases.....	50	13	16	73	15	17	24	25
Deaths.....	39	6	14	0	3	15	20	9

TABLE 7.—*Statistics on Ligation of the Common Carotid Artery*

Reference	Number of Cases	Mortality, per Cent	Cerebral Complications, per Cent	Comment
Albert, E.: Lehrbuch der Chirurgie und Operationslehre, Berlin, Urban & Schwarzenberg, 1877, vol. 1, p. 548	...	Hemorrhage, 59; aneurysm, 27; tumors, 29; neuroses, 3	25.0 Ligations for neuralgia	
Bartlett, W., and McKittrick, O. F.: Ann. Surg. **65**: 715, 1917	105 15 for secondary hemorrhage	33.0	....	Claims ligation of common carotid artery less dangerous than that of internal carotid artery
Ehrmann, J.: Des effets pro. de l'encephalie par l'obliteration des vaisseaux arteriels, Paris, J. B. Baillière et fils, 1860	213	14.0	22.0	67% of the cerebral complications fatal
Fisher: "Krank. d. Hals." Deutsche Arch. f. Chir. **34**: 55, 1880	...	56.0	32.0	
de Fourmestraux, J.: Arch. gén. d. chir. **1**: 515, 1907	71	21.0	....	
Friedlander: Inaug. Dissert., Dorpat, 1884	216	42.3	....	
Gussenbaum, cited by Walcker: Arch. f. klin. Chir. **130**: 736, 1924	...	100.0	....	
Jordan, M.: Verhandl. d. deutsch. Gesellsch. f. chir. **36**: 83, 1907	...	10.0	25.0	
Keen, W. W.: Am. J. M. Sc. **48**: 27, 1864	284	40.5	13.1	
Kranepuhl: Inaug. Dissert., Barburg, 1901	46	9.0	24.0	
Lebram, P.: Ztschr. f. Ohrenh. **50**: 176, 1905	572	....	24.5	
Ledentu, cited by Walcker	...	10.0	Less than 50.0	
LeFort, L.: Gaz. hebd. de méd. **5**: 437, 1868	370	21.0	54.0 39% of these were fatal	116 ligatures for hemorrhage; 28 patients died from secondary hemorrhage; 23 died from cerebral complications
Lestelle: Thèse de Paris, 1903-1904	126	Hemorrhage, 50; aneurysm, 27; vascular tumors of orbit, 6; 38.0	11.0	
Lewandowsky: Berl. klin. Wchnschr. **57**: 998, 1920	...	....	25.0	
Martini: Schmidt's Jahrb. **98**: 77, 1856	169	38.0	9 of 45 deaths were due to hemiplegia	
Norris, G. W.: Am. J. M. Sc. **14**: 13, 1847; cited by Pilz	151	39.0	....	
Pilz, C.: Arch. f. klin. Chir. **9**: 257, 1868	914	40.0	32.0	Hemorrhage, 220; aneurysm, 85; tumors, 137; neurologic, 33; Brasdorwardrop, 37
Pilz	228 ligations for hemorrhage	56.0	41.0	
Reis: Inaug. Dissert., Würzburg, 1885	73	42.0	23.0	Aneurysm, 32; trauma, 15; secondary hemorrhage, 10; carcinoma and sarcoma, 7; other tumors, 4; orbital, 3

TABLE 7.—*Statistics on Ligation of the Common Carotid Artery—Continued*

Reference	Number of Cases	Mortality, per Cent	Cerebral Complications, per Cent	Comment
Schlotter: Beitr. z. klin. Chir. **30**: 157, 1901	789	....	25.0	
Settler and Keller, cited by Walcker	...	4.0 to 5.0	....	
Siegrist, A.: Arch. f. Ophth. **50**: 511, 1900	172	20.3	....	
Velpeau, cited by Pilz	137	30.0	....	
Velyaminoff: Vrach **2**: 749, 1881; **3**: 490, 1882	47	2.2	12.0	Low mortality owing to preliminary daily compression of carotid 10 minutes every hour for 8 days
Wilms, in Bier, Braun and Kümmel: Chirurgische Operationslehre, Leipzig, Johann Ambrosius Barth, 1914	...	31.0	....	
Winslow, N.: Ann. Surg. **75**: 696, 1922	61 aneurysm only	25.0	12.0	Common carotid artery, 22 cases; 9 fatal; internal carotid artery, 4 cases; 1 fatal
Wyeth, J. A.: Tr. Am. M. A., 1878	789	41.0	In 7% of patients surviving	323 deaths; 7½% within 24 hours; 23% within 3 days; 45% within 7 days; 75% within 3 weeks
Zimmermann, W.: Beitr. z. klin. Chir. **8**: 364, 1891	70	31.0	25.0	

hemorrhage which we have presented. It must be remembered in studying these statistics that unless otherwise stated they include ligations for a variety of conditions, such as aneurysm, vascular tumors of the orbit, malignant processes, secondary hemorrhage, epilepsy and migraine, representing a wide range of ages. Furthermore, many of the cases in the series were collected from the preantiseptic age, and therefore the mortality would be expected to be higher than that for recent years. On examining the table more closely, one sees that the mortality varies from as low as 2.2 per cent to as high as 100 per cent. Yet, the average for the majority of the authors is between 30 and 40 per cent. (Curiously enough, Wyeth collected 33 cases in which both common carotid arteries had been ligated, with only 9 deaths, or a mortality of 27 per cent.)

In only two of the series presented has there been an analysis of cases by decades, namely, those of Pilz and Siegrist (table 5). Their figures give definite proof of the generally known fact that the mortality rises with the age, being 18 and 14 per cent for the first decade and about 50 per cent for the fourth decade. As the greatest number of ligations for hemorrhage due to suppurations occur in young patients, one would naturally expect the lowest mortality in this group. Yet, in our series there is little difference in the rates for the first four decades. The reasons for this, as we shall have occasion again to point out, is mainly that it is not so much the cerebral complication alone that kills the

patient as it is the other factors inherent in the malady itself, namely, sepsis and acute anemia.

The extremely low mortality of 2.2 per cent reported by Velyaminoff was attained by gradual compression of the artery over a period of eight days for ten minutes every hour before final ligation. The gradual or intermittent compression for the purpose of permitting the collateral circulation to develop in the brain before the ligation is completed has been investigated and experimented with by various observers during the past fifty years. Smoler used a special clamp for gradual compression of the artery. In the 7 cases in which he used this technic he did not encounter one cerebral complication. Braun discussed this phase of the subject rather fully, quoting Matjes who used an aluminum band, Hopfner, who used a metal clamp, Carrell who used a clamp and gauze band, and Smith who employed a rubber tube and clamp. Halsted used bands of fascia lata, as did Freeman. Schlotter cited Reyher, Velyaminoff, Lesser, Senger, Schoenborn and Riese as having employed the gradual preliminary compression method with excellent results. Schlotter compared Pilz' record of 13 deaths in 17 cases of ligation for resection of the jaw with his own 3 cases, in all of which recovery occurred. He claimed that temporary constriction does not predispose to thrombus formation to any greater degree than permanent ligation, and cited several experiments on animals and autopsy reports to support his contention in favor of broad temporary compression as less dangerous than ligature.

These facts are presented because they are related to the subject of cerebral complications of ligation of the carotid artery, representing an effort to minimize this danger. Naturally they are no great help to the surgeon who is confronted with the necessity of ligating the common carotid artery to control a hemorrhage that threatens the life of a patient, because then there is no time for long-continued preparatory compression, and one must take the risk of immediate ligation. Yet, should the opportunity arise when delay is not dangerous, these procedures might well be considered.

The serious cerebral complications produced by ligation of the common carotid artery are due to the sudden shutting off of the arterial supply to half the brain. It is the consensus that immediate hemiplegia is due to anemia of the brain, although there are some authors who maintain that the modus operandi of this complication has not been definitely proved. Grüber and Werner stated that "we are still in the dark as to the modus of development of brain disturbances following interruption of the blood stream on one side, and all previous attempts to circumvent this danger are but gestures in the dark." Perthes described autopsies which showed a free communication of the vessels on the ligated side with those on the other side through a normal circle

of Willis; this failed to explain the phenomenon. Walcker stated that there are two kinds of circle of Willis, open and closed, depending on the presence of communicating arteries. The first is seen in mesaticephalic and brachycephalic persons, and the second in dolichocephalic persons. Walcker claimed that ligation is less likely to be followed by cerebral disturbance in the former than the latter. Further, he believes that one reason why the incidence of complications is higher in adults than in children is because the surface vessels in adults are more tortuous and therefore offer more resistance to the blood stream, thus favoring thrombosis. Another point which he as well as other authors stress is the desirability of ligating the internal jugular vein at the same time if the jugular vein is large. Grüber and Werner claimed that ligation of the jugular vein does not help as much as was thought. Kosterlitz expressed the same view. Boeck, on the other hand, stated that when the arterial supply is cut off venous stasis occurs because of lack of pressure and this leads to paralysis. Nevertheless, most of the authors are in practical agreement that early hemiplegia is the result of anemia of the brain, and that this complication offers a better prognosis than delayed hemiplegia, which they believe is due to thrombosis or an embolus which leads to softening. Brüning expressed the belief that of the three pathologic entities, namely, acute anemia of the brain, thrombus or embolus with softening and venous stasis with edema, the last named is the least serious and offers a fairly good prognosis for gradual recovery.

As for combating these serious complications, we have already mentioned gradual compression and delayed ligation in connection with preventing the acute anemia, a desirable procedure when time will permit. Also we have pointed out the advisability of tying the internal jugular vein, as suggested by Walcker. We might further add in connection with the question of anemia that ligation of the common carotid artery is said to be less likely to be followed by acute anemia than ligation of the internal carotid artery because there must be some back flow from the collateral circulation of the external carotid artery around the bifurcation into the internal carotid artery. On the other hand, this procedure has been known to cause secondary hemorrhage after ligation of the common carotid artery, so that it was necessary to ligate the external carotid artery as well. One cannot, therefore, expect to reap the beneficial effects of a back flow by way of the external carotid artery into the internal carotid artery without running the risks of hemorrhage from the erosion or a ruptured aneurysmal sac. There is no objection to tying the internal carotid artery instead of the common carotid artery except that in many instances the pathologic process is so extensive as to render exposure of the bifurcation very difficult, if not dangerous. (The few ligations of the internal carotid artery which

we noted in the case histories were included under the heading of "Ligation of the Common Carotid Artery"). As for prevention of thrombosis, it has been pointed out by many authors that injury to the intima through the firm ligation is to be considered the primary cause. Freeman stated that the intima is and may be easily ruptured in the application of a ligature, and he therefore advoated using a band of fascia lata which will constrict the vessel without injuring it. Certainly there have been a sufficient number of cases of delayed hemiplegia to lend color to this hypothesis, and it is therefore worthy of consideration.

The importance of hemiplegia and other cranial symptoms in the prognosis following ligation may be readily seen in the table of 520 ligations by Pilz in which the mortality is considered in connection with each symptom separately (table 6). In 73 cases in which only vertigo, headache and transient loss of consciousness occurred there were no fatalities; in 15 cases of sensory disturbances there were 3 deaths. On the other hand, 50 cases of hemiplegia resulted in 38 deaths; 17 cases of coma yielded 15 deaths, and 24 cases of delirium were fatal.

The occurrence of ocular complications was studied by Siegrist, who found 4 per cent permanent impairment in a series of 997 cases. Otitic symptoms are still less frequent, occurring, according to Lebram, in only 0.6 per cent of 1,200 cases.

Nevertheless, even though the mortality from cerebral complications is high, it does not account for the generally high mortality from ligature of the common carotid artery because, as has been shown, cerebral complications of all kinds occur in only 25 per cent of all ligations for all ages combined. There are other causes of death which cannot be attributed solely to the effects of ligation. Siegrist (table 3) analyzed 321 fatalities and found 20 per cent due to sepsis, 17 per cent to secondary hemorrhages, 11 per cent to severe constitutional disturbances, 22 per cent to cranial complications and 26 per cent to causes not definitely established. Wyeth analyzed 200 deaths and found 22 per cent due to secondary hemorrhage, 11 per cent to exhaustion, 34 per cent to cranial complications and 11 per cent to hemorrhage plus other causes. Siegrist stated that death in most cases is due to (1) the fatal maladies which led to the ligation; (2) intercurrent maladies following ligation but unrelated to it; (3) sepsis, and (4) secondary hemorrhage due to infection. A striking proof of these facts may be found in 40 ligations for epilepsy collected by Wyeth in which the patients were not debilitated by hemorrhage, pain or infection; there were only 2 deaths. Certainly this tends to show that the high mortality from ligation of the common carotid artery cannot be ascribed entirely to the cerebral complications and that other constitutional factors inherent in the ailments themselves must share in the blame.

Author	Sex and Age of Patient	Infection	Evacuation; Spontaneous or by Incision	Hemorrhage; Spontaneous or Following Incision	Hemorrhage; Single or Recurrent	Ligation of Major Vessel	Disturbance Due to Ligation, If Any	Result	Autopsy Report and Comments
Adolphus, E.: Edinburgh M. & S. J. **59**: 104, 1843	M 30	Tonsillar; cervical	Incision in neck ninth day; sloughing sixteenth day	Hemorrhage twenty-third day, 72 ounces	Recurrent for 2 weeks; total loss, 560 ounces; also bled from mouth	No ligation; only palliative treatment	.....	Recovery	Bleeding traced to inferior thyroid artery
Alford, in Liston, Robert: Practical Surgery, London, J. Churchill, 1846, p. 189	F 32	Sore throat; cervical abscess	Spontaneous over sternocleidomastoid muscle; also over sternum	Spontaneous from sloughing ulcer of neck	Recurrent for several days	.....	.....	Death	Large erosion of internal jugular vein
Aubriot: Ann. d. mal. de l'oreille, du larynx **41**: 508, 192..	Not given	No pus	.....	Spontaneous from tonsil	Recurrent	Tonsils removed and bleeding vessels ligated	None	Recovery	
Aubriot	Not given	No pus	.....	Spontaneous from tonsil	Recurrent	.....	None	Recovery	
Baader, A.: Cor.-Bl. f. schweiz. Aerzte **5**: 616, 1875	F 4	Scarlet fever; cervical suppuration; bilateral parapharyngeal	Incision on one side; recovery; swelling of opposite side; incision; bloody coagulum	Followed incision of opposite side; temporary abatement from bandage and pack	Fatal recurrence from throat 3 days later	.....	.....	Death	Large abscess cavity from internal to carotid artery; erosion of external carotid artery; erosion of abscess into throat; spurious aneurysm
Baader	F 6	Scarlet fever; dense phlegmon of neck on tenth day; extensive sepsis	Incision; putrid bloody pus	Followed incision	Single	.....	.....	Death	Extensive phlegmon of all deep structures of neck to sternum; erosion of internal jugular vein
Baader	? 3	Scarlet fever; exudate in throat; extensive discharge from ear	.....	Spontaneous from ear twelfth day	Fatal recurrence 2 days later	.....	.....	Death	Necrosis of posterior wall of middle ear; erosion of transverse sinus
Babbit, J. A.: Laryngoscope **34**: 37, 1924	M 7	Retropharyngeal; swelling of neck	.....	Spontaneous	Single	.....	.....	Death	Erosion of tributary vessels; carotid artery intact
Barret: Lancet **1**: 287, 1847	F 5	Scarlet fever; cervical phlegmon; sloughing of all tissues	No pus mentioned	Spontaneous	Recurrent	.....	.....	Death	Erosion of internal jugular vein; sheath of carotid artery invaded; tissues of neck gangrenous from parotid to clavicle
Beyme: Deutsche Ztschr. f. Chir. **140**: 309, 1917	M ?	Retropharyngeal	Incision	Five days after incision	Recurred next day; fatal	.....	.....	Death	Erosion of internal carotid artery, perhaps from rubber drainage tube

Billroth: Chirurgische Erfahrungen, Arch. f. klin. Chir. **10**: 157, 1869	M 52	Cellulitis of neck	Spontaneous rupture of abscess into parapharyngeal space	Sudden profuse hemorrhage into throat; rapidly fatal	Single	Hemorrhage too sudden for therapy	.....	Death	Erosion of left superior thyroid artery
Bittner: Prag. med. Wchnschr. **32**: 405, 1898	? 15 weeks	Retropharyngeal	Incision	Fatal hemorrhage 1 day after incision	Single	.....	.....	Death	Perforation of common carotid artery, believed to be due to erosion from drainage tube
Bloch: Soc. d. larygn. d. hôp. de Paris **10**: 11, 1926	F	Peritonsillar	Incision	Two days after incision	Single; foudroyant	External carotid artery	None	Recovery	
Bloxam, W.: Med.-Chir. Tr., London **26**: 112, 1843	? 5	Scarlet fever 3 weeks; suppuration of glands of neck	Spontaneous	Hemorrhage fifth day after incision	Continuous for 3 days	.....	.....	Death	Erosion of internal jugular vein
Bobone, T.: Boll. d. mal. d. orecchio, d. gola, d. naso **18**: 217, 1905	F 19	Peritonsillar	Incision sixth day	Followed incision in 24 hours	Continuous for several hours	.....	.....	Recovery	
Bourget: Gaz. d. hôp. **48**: 873, 1875	M 20	Swelling of right side of throat	Presence of pus presumed but not proved	Spontaneous	Recurrent	None	.....	Death 15 days from onset	No autopsy; source of hemorrhage probably internal carotid artery
Brauer: Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**: 561 1933	M 5	Peritonsillar; otitis media; erysipelas; parapharyngeal	Incision, repeated 8 days later	Followed second incision	Recurrent; next day cyanosis; tracheotomy	.....	.....	Death	Blood-filled cavity in parapharyngeal space; external maxillary artery broken down
Brauer	F 9	Peritonsillar; cervical glands	Incision eleventh day	Spontaneous from mouth, nose and ear eighth day	Recurrent following incision	Incision of sternocleidomastoid muscle; bleeding vessel not located; cavity packed	Horner's syndrome and hypoglossal paresis followed operation	Recovery	Tampons used in wounds in throat and neck for 10 days; 2½ years later Horner's syndrome still present but hypoglossal nerve recovered
Brauer	M 50	Peritonsillar	Spontaneous eighth day; incision eleventh day	Spontaneous eighth day; tampon	Recurrent; bleeder located at base of tonsil; tonsil removed; vessel tied	.....	.....	Recovery	
Brauer	M 46	Peritonsillar	Spontaneous second day	Spontaneous	Recurrent on eighth day; bleeder located at lower pole; ligated	.....	.....	Recovery	
Brauer	M 21	Peritonsillar	Incision second day	Followed incision; tampon	Recurred when tampon was removed; controlled by tampon	.....	.....	Recovery	
Brauer	M 27	Peritonsillar 5 days	Incised twice; spread 4 times	Followed incision	Recurrent; controlled by tampon	.....	.....	Recovery	

Peritonsillar, Retropharyngeal, Parapharyngeal and Cervical Abscesses with Hemorrhage—Continued

Author	Sex and Age of Patient	Infection	Evacuation; Spontaneous or by Incision	Hemorrhage; Spontaneous or Following Incision	Hemorrhage; Single or Recurrent	Ligation of Major Vessel	Disturbance Due to Ligation, If Any	Result	Autopsy Report and Comments
Brauer	M 48	Peritonsillar	Incision	Followed incision	Recurred 4 days later; controlled by tampon	.....	.....	Recovery	
Brauer	F 15	Peritonsillar	Incision tenth day	Followed incision	Recurrent; controlled by ice compresses	.....	.....	Recovery	
Brauer	M 24	Peritonsillar	Incision eighth day	Followed incision 2 days	Single	.....	.....	Recovery	
Brewer: Yale M. J. **5**: 88, 1898	M 25	Posterior surface of soft palate	Spontaneous	Spontaneous following rupture of abscess	Recurrent	No ligation; palliative local pressure with sponges; only temporary effect	.....	Death from hemorrhage	No autopsy; vessel involved not determined
Brill: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **11**: 810, 1928	M 28	Peritonsillar	Spontaneous after 5 days	Six hours after rupture of abscess	Recurrence 1½ days after first hemorrhage	No ligation; cavity packed at first	.....	Recovery	
Brown: A. M. J., London **2**: 991, 1854	F 7½	Scarlet fever; cervical	Incision seventeenth day	Followed incision 3 days; ¾ pint from nose and throat	Recurred from nose and throat 2 days later, 1 pint; fatal hemorrhage 2 days later, 1 quart	.....	.....	Death	Ulcerated opening in pharynx leading into sac external to superior constrictor; not certain whether internal carotid artery was eroded
Buev: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **13**: 184, 1928	Not given	Peritonsillar	Not given	Spontaneous	Single	No ligation	.....	Death	Internal carotid artery
Caboche and Borst: Ann. d. mal. de l'oreille, du larynx **44**: 850, 1925	? 21	Peritonsillar	Incision sixth day	Hemorrhage 2 hours after incision of abscess	Recurrent second and tenth days	No ligation	.....	Recovery followed incision of abscess that developed in neck by tenth day	
Caboche and Borst.....	? 40	Peritonsillar	.....	Daily spontaneous bleeding from above upper pole of tonsil	Recurrent	Preparations made; no chance to complete ligation	.....	Death	None
Caboche and Borst.....	F 9	No pus	.....	Spontaneous third day	Recurred on fifth day	.....	.....	Death on sixth day	None
Carmichael, cited by Lebram from Vergely: Ztschr. f. Ohrenh. **51**: 4, 1906	? 5 wks.	Retropharyngeal	.....	.....	Single	.....	.....	Death	Retropharyngeal erosion of branch of external carotid artery

Carmody: Colorado Med. **12**: 261, 1915	M 3	Peritonsillar	Spontaneous ninth day	Spontaneous ninth day; opening seen at upper pole of anterior pillars	Recurrent oozing 18 hours later; a few hours later fatal hemorrhage	.....	.....	Death	
Carteaux: J. de méd. et chir. prat., October, 1845	? 20 mos.	Measles; cervical bilateral	Incision of both sides	Followed incision, 4 days	Not stated	.....	.....	Death	Erosion of internal jugular vein
Cayton, cited by Lebram from Vergely: Ztschr. f. Ohrenh. **51**: 4, 1906	M 38	Peritonsillar	Spontaneous eighth day	Spontaneous, simultaneous with opening of abscess	Single	.....	.....	Death	Erosion of internal carotid artery
Chappell: Tr. Am. Laryng. A., 1900, p. 213	M 27	Peritonsillar; 5 weeks	Incisions (4)	Following last incision, 4 days	Recurrent	.....	.....	Recovery	
Chassaignac: Deutsche Ztschr. f. Chir. **57**: 591, 1902	M 26	Retropharyngeal; cervical	Incision	Immediately after incision	Single	Common carotid artery	Aphonia; severe headache	Recovery	
Clayton, cited by Chappell	..	Peritonsillar	Spontaneous	Several hours after incision	Single	.....	.....	Death	Erosion of internal carotid artery
Clutton, H.: Brit. M. J. **1**: 395, 1897	M 28	Peritonsillar	Incision 3 days after first hemorrhage	Profuse; spontaneous	Single	Common carotid artery	.....	Recovery	
Coffin, L. A.: Ann. Otol., Rhin. & Laryng. **20**: 482, 1911	F 21 mos.	Peritonsillar; swelling of angle of jaw	Spontaneous; recurrence and incision 2 days later, but no pus	Hemorrhages from ear before abscess opened; fatal hemorrhage 3 days after incision	Single	.....	.....	Death	
Cohen, D.; Cohen, A. E., and Miller, A. J.: Internat. J. Med. & Surg. **45**: 120, 1932	F 12	Retropharyngeal	Incision; no pus on seventh day	Severe hemorrhage day after incision; pupils dilated	Recurrent next week; fatal hemorrhage 3 days later	.....	.....	Death	Erosion of internal carotid artery 2 cm. from skull; false aneurysm; old tuberculous infection in lungs; probable tuberculous gland of pharynx at onset of abscess
Craigie, D.: New York M. J. **48**: 233, 1888	M 45	Retropharyngeal 17 days	Spontaneous	Immediate; spontaneous	.....	.....	.....	Death	Slough seen in postpharyngeal wall and tonsil; small arteries exposed; likely twigs of ascending pharyngeal vein
Crisp, Edwards: A Treatise on the Structure, Disease and Injuries of the Blood Vessels, London, J. Churchill, 1847, p. 282	..	Probably retropharyngeal	.....	Spontaneous; fatal	.....	.....	.....	Death	Specimen in the Chatham Museum described, showing ulceration of internal carotid artery caused by abscess of base of skull with fatal hemorrhage into pharynx

Peritonsillar, Retropharyngeal, Parapharyngeal and Cervical Abscesses with Hemorrhage—Continued

Author	Sex and Age of Patient	Infection	Evacuation; Spontaneous or by Incision	Hemorrhage; Spontaneous or Following Incision	Hemorrhage; Single or Recurrent	Ligation of Major Vessel	Disturbance Due to Ligation, If Any	Result	Autopsy Report and Comments
Critchett: Lancet **1**:664, 1854	M 28	Neck	Incision; external tenth day; spontaneous in throat	Five days after incision	.....	Common carotid artery	None	Death	
David: Lancet **1**:241, 1847	M 6	Scarlet fever; swelling in neck 2 weeks	Incision; foul pus; spontaneous exposure of internal jugular vein	Sudden, fatal hemorrhage 2 days later	Single	.....	.....	Death	
De Bal: Dublin J. M. Sc. **1**:533, 1846	F 12	Scarlet fever; cervical	Incision of abscess of neck	Hemorrhage seventh day after incision	Recurrence same day; fatal	.....	.....	Death	Erosion of internal jugular vein
Deperet: Union méd. **5**: 414, 1850	M 14	Scarlet fever; peritonsillar abscess; dyspnea	Emetic caused spontaneous rupture of abscess; later incision of abscess of parotid area	Hemorrhage 3 days after incision	Recurrent; controlled by pressure probably on internal jugular vein	.....	.....	Recovery	
Desgouttes: Zentralbl. f. Chir. **37**:998, 1910	?	Neck; broken down lymph gland (carcinoma)	.....	Spontaneous	Single	Common carotid artery	None	Recovery	
Dolbeau: M. Times & Hosp. Gaz. **1**:487, 1864	F 16	Cervical glands	Spontaneous; incision after hemorrhage	Spontaneous seventeenth day	Single	External carotid artery	None	Recovery	
Duke: Lancet **1**:233, 1848	M 32	Pulsating tonsillar region	Suspected aneurysm; no incision	.....	.....	Common carotid artery	None	Death followed 4 weeks after ligature due to a debauch	Injury to head 6 months previously, sore throat; pulsating mass disclosed; soft and elastic; rôle of infection doubtful
Duncan, J.: Edinburgh M. & S. J. **62**:117, 1844	M 60	"Swelling in throat"; pus doubtful	.....	Spontaneous	Recurrent	Common carotid artery	Continuous bleeding fourth, tenth eleventh and twelfth days	Death twelfth day	
Dunn, T. D.: Univ. M. Mag. **3**:455, 1891	? 3½	Peritonsillar	.....	Spontaneous (?)	.....	Common carotid artery	No further information	Recovery	
Ehrmann: Bull. et mém. Soc. de Chir. de Paris **4**: 664, 1878	M 22	Swelling of angle of jaw; dysphagia	Spontaneous	Spontaneous; severe	Recurrent few hours later	Common carotid artery	None	Recovery	

Else, in Medical Observations and Inquiries, ed. 2, London, T. Cadell, 1769, vol. 3, p. 177		Specimen of erosion between exit of right and left carotid arteries; death due to suffocation from pressure on trachea							
Erichsen: Deutsche Ztschr. f. Chir. **14**: 1, 1881	..	Phlegmonous angina	Spontaneous into pharynx	Spontaneous 7 days later	Single	.....	.....	Death	Autopsy: erosion of internal carotid artery
Felheisen: Deutsche Ztschr. f. Chir. **14**: 1, 1881	M 34	Parapharyngeal; peritonsillar	Incision seventh day	Immediately after incision; tracheotomy	.....	.....	.....	Death	Autopsy: false aneurysm of common carotid artery with erosion
Frank, Ira: Retropharyngeal Abscess, J. A. M. A. **77**: 517 (Aug. 13) 1921	..	Retropharyngeal	Incision	Immediate	Single (?)	.....	.....	Death	No further details given
Frank	..	Retropharyngeal	Incision	Followed incision	Single (?)	.....	.....	Death	
Franklin, M. M.: New York M. News **72**: 237, 1898	M 8	Retropharyngeal; pulsating swelling in neck	.....	Spontaneous; mouth and nose	Recurrent	.....	.....	Recovery in 1 year	Aphasia and hemiplegia on left day after first hemorrhage
Galloway, T. C.: Ann. Otol., Rhin. & Laryng. **35**: 1278, 1926	F 11	Retropharyngeal	Incision	Bleeding 12 hours after incision	Recurrent five and eight days later	Internal carotid artery	.....	Death	Erosion of internal carotid artery near base of skull
Gerlings, P. G.: Acta otolaryng. **17**: 394, 1932	8	Throat; peritonsillar and parapharyngeal; rupture into auditory canal	Spontaneous	Spontaneous accompanying	Recurrent and swelling in neck; miosis	.....	.....	Recovery after operation on mastoid	Swelling simulating Bezold's mastoiditis, ptosis and miosis preceded operation; lasted one month
Gerlings	6	Sore throat 3 weeks; peritonsillar abscess; homolateral miosis	Spontaneous; swelling of neck persisted	Spontaneous	Recurrent for days; fatal hemorrhage after puncture of neck	.....	.....	Death after fulminating hemorrhage	
Gerlings	? 8	Scarlet fever; peritonsillar; swelling in neck; miosis	.....	Spontaneous	Single	.....	.....	Death after first and only hemorrhage	Erosion of external carotid artery
Gleitsmann: New York Med. Monatschr. **1**: 652 1889	M 24	Peritonsillar; parapharyngeal	Incision tenth day; no pus; pus on thirteenth day	After second incision	Recurred 8 days later; again packed	.....	.....	Recovery	
Gordon: Dublin M. Press **15**: 242, 1846	..	Scarlet fever	.....	.....	.....	.....	.....	Death	No details given; fatal hemorrhage from erosion of internal jugular vein
Greenfield, S. D.: Peritonsillar Infection, Arch. Otolaryng. **7**: 239 (March) 1928	..	Peritonsillar	.....	Spontaneous	Recurrent	.....	.....	Recovery	
Grisolle (1865), cited by Newcomb from Vergely: Tr. Am. Laryng. A., 1908	..	Throat	.....	Spontaneous	Single	.....	.....	Death	Erosion of internal carotid artery

Peritonsillar, Retropharyngeal, Parapharyngeal and Cervical Abscesses with Hemorrhage—Continued

Author	Sex and Age of Patient	Infection	Evacuation; Spontaneous or by Incision	Hemorrhage; Spontaneous or Following Incision	Hemorrhage; Single or Recurrent	Ligation of Major Vessel	Disturbance Due to Ligation, If Any	Result	Autopsy Report and Comments
Gross, S. W.: Am. J. M. Sc. **122**: 337, 1871	M 21 mos.	Scarlet fever; swelling of angle of jaw	Incision; pus	Fatal hemorrhage 4 days later	Single	.....	.....	Death	Deep widespread gangrene of neck; erosion of internal jugular vein
Guttman: Personal communication to Salinger	M 5½	Retropharyngeal; swelling of neck	.....	Spontaneous	Recurrent	Common carotid artery	Contralateral facial paresis and hemiplegia preceded ligation by 2 days; disappeared later	Recovery	
Hale: London J. Med. **2**: 720, 1850	M 8	Scarlet fever; sloughing ulceration of uvula and tonsils; subclavian abscess	Incision	Several days after incision	Recurrent when packs were released	.....	.....	Death	Abscess cavity from below clavicle to axilla and up into neck to angle of jaw; erosion of subclavian and internal jugular veins
Hall, R. W.: Boston M. & S. J. **117**: 604, 1887	M 20	Peritonsillar 2 weeks prior to examination	Spontaneous	Spontaneous; numerous and recurrent	Recurrent over a period of 11 days	.....	.....	Death followed sudden fulminating hemorrhage	Fatal hemorrhage so terrific it could come only from vessel size of internal carotid artery
Hargrave: Dublin Quart. J. **8**: 80, 1849	M 61	Erysipelas of neck; infection	Incision; recurrence of abscess	Followed incision	Recurrent	.....	.....	Death	Patient refused further treatment
Heaton, G.: Birmingham M. Rev. **30**: 304, 1891	M 20	Peritonsillar 10 days; parapharyngeal	Spontaneous	Spontaneous from nose and throat	Recurrence from ear, nose and throat for several days; fatal bleeding	Tracheotomy; ligation of common carotid artery (loose loop)	Fatal hemorrhage 2 days after loop put around carotid	Death	External carotid artery normal; infiltration about internal carotid artery, same eroded and ruptured
Heindl, A.: Wien. med. Wchnschr. **81**: 1572, 1931	..	Peritonsillar	Incision	After incision	Continuous	.....	.....	Recovery following enucleation of tonsil and ligation of ascending pharyngeal artery	
Heindl	..	Peritonsillar	Incision	Spontaneous	Recurrent	.....	.....	Recovery following injection of 10 cc. of a hemostatic preparation	

Hinsberg, K.: Folia oto-laryng. **34**: 375, 1931	Child	Peritonsillar	Incision	Immediate	Single	.....	.....	Death	False aneurysm
Hinsberg	Child	Peritonsillar	Incision	Immediate	Single	Common carotid artery	.....	Recovery	False aneurysm (?)
Hinsberg	Child	Peritonsillar	No therapy	Spontaneous	Not stated	.....	.....	Death	False aneurysm
Hodgson and Hargrave: Lancet **1**: 866, 1841		Three cases of aneurysm in neck; most likely of carotid artery; details lacking							
Hoffman: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **12**: 128, 1928	7 mos.	Retropharyngeal and swelling of neck	.....	Spontaneous into auditory canal	Recurrent	.....	.....	Death	Small aneurysm of internal carotid artery; erosion into abscess; rupture into middle ear
Hoffman	M 2	Retropharyngeal	Spontaneous	Spontaneous	.....	.....	.....	Death in a few days	Erosion of internal carotid artery
Hoffman: London & Edinburgh. Monthly J. M. Sc. **4**: 632, 1844	? 5	Scarlet fever; cervical palpatory thrill and auscultatory bruit	Puncture	Immediate with puncture	Single	.....	.....	Death	Internal jugular vein perforated like a sieve; distance, ¾ inch (1.87 cm.)
Hopkins: Tr. Am. Laryng. A. **38**: 105, 1913	M 26	Peritonsillar	Incision; no pus	Hemorrhage 2 days later; controlled by pressure	Recurrence next day fatal	.....	.....	Death	Autopsy refused; examination of throat showed rupture through posterior pillar; perhaps of carotid artery
Horgan, J. B.: J. Laryng. & Otol. **46**: 335, 1931	M 37	Peritonsillar	Incision	Followed incision	Recurrent	.....	.....	Recovery; incision; large clots removed; cavity (pharyngeal) packed	
Horner, A.: Prag. med. Wchnschr. **39**: 365, 1914	M 57	Lateral part of pharynx	Incision	Three weeks after incision	Recurrent	Common carotid artery; erosion found	Hemiplegia and aphasia; coma	Recovery in 5 months	
Huber: Arch. f. klin. Med. **8**: 422, 1871	? 6	Scarlet fever; cervical 2 weeks later	Incision; no pus; enlarged by finger	Followed probing with finger; fatal	Single	.....	.....	Death	
Hughes: Dublin M. Press **15**: 241, 1846	? 6	Scarlet fever; abscess of throat	Spontaneous	Spontaneous tenth day	Single	Common carotid artery	.....	Death from exhaustion	
Hulbert: St. Louis Courier Med. **16**: 265, 1886	F 19	Scarlet fever; pharyngitis; swelling of parotid region	Recurrence of peritonsillar swelling 2 weeks later; incision; no pus; blood; cervical swelling	Spontaneous from area near tonsil	Recurrence 2 weeks later from incision of peritonsillar swelling	Patient died on table while being prepared for ligation	.....	Death	Rupture of internal carotid artery
Hutchinson: J. Laryng. **34**: 122, 1919	F 16	Peritonsillar	Incision	Followed incision	Two recurrences	.....	.....	Death 2 days later; severe sepsis	None

Peritonsillar, Retropharyngeal, Parapharyngeal and Cervical Abscesses with Hemorrhage—Continued

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Hynes: Lancet **2**: 431, 1870	M 4	Postscarlatinal; cervical swelling	No pus	Spontaneous from ear and throat twenty-sixth day	Recurrent 24 hours	.....	.....	Death	Internal carotid artery eroded into ear and drained partly through eustachian tube
Immerman, cited by Lebram from Vergely: Ztschr. f. Ohrenh. **51**: 4, 1906	17	Scarlet fever; peritonsillar and parapharyngeal	.....	Spontaneous from mouth and nose eighth day	Single; fatal in 5 minutes	.....	.....	Death	Erosion of carotid artery (?)
Jager: Folia oto-laryng. **12**: 369, 1924	F 45	Grip 4 weeks; sore throat; swelling external part of neck	Incision, external; large abscess extending back to spine	Spontaneous from wound 6 days after opening of abscess	Single	Internal carotid artery	Hemiplegia	Death next day	None
Jastram, M.: Beitr. z. klin. Chir. **93**: 341, 1914	F 54	Swelling; peritonsillar and parapharyngeal 4 weeks; no pulsation	Incision; no pus	Immediate on incision	Recurred after ligation of carotid artery	External carotid artery	Hemorrhage followed; partly controlled by clamp	Death	Aneurysm of external carotid artery; absence of pulsation attributed to thick walls and lining thrombi
Johnson, W. B.: Tr. Am. Laryng., Rhin. & Otol. Soc. **7**: 226, 1901	M 4	Peritonsillar 10 days	Attempt to open unsuccessful	Spontaneous from ear; swelling increased	Recurrent after several days from nose, ear and throat	.....	.....	Death	Probable aneurysm of internal carotid artery
Kaplan, Y. B.: Russk. Vrach **5**: 1616 (Dec. 23) 1906	M 19	Parapharyngeal	Incision; external; cyanosis; choking; tracheotomy	Followed incision by 48 hours (from wound)	Single	Common carotid artery	Comatose	Death next day	Erosion of internal carotid artery
Kaplan	M 22	Parapharyngeal (from tonsillitis)	Incision external	Followed incision by 13 days (from wound)	Single	Common carotid artery	Paralysis of opposite arm and leg; improved after 10 days	Death (convulsions)	Hemorrhagic pleurisy
Kaufman, A.: Laryngoscope **41**: 826 (Dec.) 1931	F 2	Retropharyngeal	Not opened; rupture into auditory canal	Spontaneous first from ear; then nose, and 5 days later from throat (fatal)	Recurrent	.....	.....	Death	False aneurysm of internal carotid artery; erosion of branch that goes to tympanic cavity
King, K.: London & Edinburgh Monthly J. M. Sc. **27**: 177, 1843	M 4	Scarlet fever; parotid and cervical	Spontaneous	Spontaneous 3 days later	Recurred same day; mass increased in size; recurred when tampon was removed	.....	.....	Death	One sac below digastric and parotid; other sac external to parotid gland and carotid arteries; external wall of internal jugular vein sloughed near base of skull

Klarfeld: Wien. klin. Wchnschr. **28**: 1361, 1915	? 18	Parapharyngeal	Sepsis	Spontaneous from throat	Single	Common carotid artery	.....	Death 12 hours later	Aneurysm and erosion of internal carotid artery
Klug: Zentralbl. f. Ohrenh. **2**: 383, 1920	?	Retropharyngeal from otitis media and petrous caries	.....	Spontaneous; fulminating	Single	.....	.....	Death	Erosion of internal carotid artery just below entrance into skull
Koch, J.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**: 245, 1932	M 33	Peritonsillar swelling behind and below tonsils; pressure sensation in nose and throat	Spontaneous	Spontaneous with vomiting	Recurrent for 2 days with relief of pressure sensation; pulsation on palpation	Ascending pharyngeal and ascending palatine veins; thrombosed veins resected	Hemorrhage of lower pole of tonsil next day; tonsil removed; vessel tied	Recovery	
Lange, W.: Charité-Ann. **32**: 457, 1908	F 14	Peritonsillar and parapharyngeal	Incision of peritonsillar abscess; no pus	Spontaneous from ear	Recurrent	External carotid artery; glands found suppurating	Paralysis of hypoglossal nerve	Recovery	
Lebram, P.: Ztschr. f. Ohrenh. **51**: 4, 1906	M 2	Retropharyngeal and submaxillary	Submaxillary incision negative; peritonsillar incision	Fatal hemorrhage followed peritonsillar incision	Single	.....	.....	Death	None
Lebram	F 9	Peritonsillar	Incision	Spontaneous	Recurrent for 3 days preceding incision of abscess	Common carotid artery and internal jugular vein	Hemiplegia; disappeared rapidly	Recovery	
Le Fort: Bull. Soc. de chir. **2**: 282, 1870; cited by Vergely and Mery	?	Phlegmonous angina	.....	Spontaneous; fulminating	.....	.....	.....	Death	Erosion of internal carotid artery
Le Mesurier, A. B.: Canad. M. A. J. **25**: 551, 1931	F 18 mos.	Retropharyngeal; swelling of neck also	.....	Spontaneous from mouth	Recurred 10 hours later; fatal	.....	.....	Death	Long, jagged erosion of internal carotid artery ½ inch (1.27 cm.) from base of skull; false aneurysm communicating with artery and pharynx
Le Mesurier	F 14 mos.	Retropharyngeal; both sides of neck swollen	Spontaneous; first from ear; incision of throat fifth day; no pus	Spontaneous from ear first	Hemorrhage from throat 6 days after incision; fatal	.....	.....	Death	Erosion of internal carotid artery 1 inch (2.5 cm.) above bifurcation; false aneurysm with rupture into pharynx; also rupture into middle ear
Le Mesurier	F 17 mos.	Retropharyngeal; neck swollen	.....	Spontaneous	Recurrent; three severe hemorrhages; transfusion; hemorrhage again	Internal carotid artery; transfusion	Immediate hemiplegia of arm; recovered completely; leg still spastic one year later	Recovery	

Peritonsillar, Retropharyngeal, Parapharyngeal and Cervical Abscesses with Hemorrhage—Continued

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Le Mesurier	M 2½	Retropharyngeal; neck swollen	.....	Spontaneous tenth day	Recurrent smaller hemorrhages; opening seen behind and below tonsil	Internal carotid artery, above erosion that was seen; transfusion	Apnea immediate	Death 12 hours later	No autopsy
Le Mesurier	F 10 mos.	Retropharyngeal; swelling of both sides of neck	Profuse foul discharge from ear	Spontaneous	Recurrent; transfusion; bleeding from ear, nose and throat	Internal carotid artery; hemorrhage ceased; recurred from wound 15 days later	Severe infection of neck; profuse hemorrhage from wound 15 days later; ligation of common carotid artery; bleeding ceased	Recovery	
Le Mesurier	F 8	Retropharyngeal; swelling of neck; hemolytic streptococci	Incision; no pus; incision of neck later	Spontaneous seventh day	Recurrent next day; profuse hemorrhage from ear; transfusion	.....	.....	Death	Erosion of internal carotid artery ½ inch (1.27 cm.) below base of skull; false aneurysm; ruptured into pharynx and through auditory canal
Le Mesurier	F 9	Retropharyngeal; swelling of neck	Incision tenth day; hemolytic streptococci	Severe 2 days after incision; opening seen behind tonsil	Single	Common carotid artery; transfusion	Weakness of arm and face; cleared in a few hours; wound of throat fungating; fatal hemorrhage 10 days later	Death	No autopsy; fatal hemorrhage probably came from internal carotid artery by back flow from external carotid artery
Le Mesurier	M 14 mos.	Retropharyngeal; swelling of neck 3 weeks later	Incision; no pus; severe hemorrhage	Immediate on incision	Single	Common carotid artery; also internal carotid artery; transfusion	No signs of cerebral disturbance	Death 3 days after ligation from sepsis	No autopsy
Liang, P. T.; Liang, P. K., and Lei, C. H.: Brit. M. J. **1**: 967, 1925	M 24	Retropharyngeal	Incision; dysphagia; tracheotomy	Severe hemorrhage seventh day	Single	Common carotid artery	.....	Recovery	
Lidell: Am. J. M. Sc. **86**: 321, 1883	M 15	Retropharyngeal	Spontaneous eighteenth day	Spontaneous	Recurrent for 10 days from mouth and nose	.....	.....	Death	Abscess cavity surrounded branches of carotid artery; immediate source of hemorrhage not determined

Lifschutz, J.: Fatal Hemorrhage Resulting from Extension of a Retropharyngeal Abscess to Deep Vessels of Neck, Arch. Otolaryng. **14**: 149 (Aug.) 1931	M 3	Retropharyngeal 5 days	.....	Spontaneous fatal hemorrhage on way to hospital	Single	.....	.....	Death; author believes internal carotid artery eroded	
Liston, cited by Fehleisen: Deutsche Ztschr. f. Chir. **14**: 1, 1881	M 9	Swelling of neck and pharynx; slow development (2 months)	Incision	Immediate on incision	Single	Common carotid artery	.....	Death 15 days later	False aneurysm of internal carotid artery; near bifurcation
Long: Sajous Univ. Ann. M. Sc. **3**: 289, 1888	?	Peritonsillar	Incision	Immediate on incision	Single; patient choked; tracheotomy	.....	.....	Death	
Loounis: Laryngoscope **43**: 70, 1933; discussion on Constans	Child	Retropharyngeal	Incision	Spontaneous	Single	.....	.....	Death	Incision into anomalous internal carotid artery
Loose: Deutsche Ztschr. f. Chir. **140**: 309, 1917	..	Retropharyngeal	Incision by Burkhard method	Five days after incision; fatal	Single	.....	.....	Death	
Loose	..	Retropharyngeal	Incision by Burkhard method	Four days after incision	.....	.....	.....	Death	
Lorance: Personal communication to Salinger by Nash	F 4	Peritonsillar	Incision	Hemorrhage 10 days after incision	Recurred later; fatal	.....	.....	Death	
Lovegrove: Lancet **1**: 729, 1870	Male child	Postscarlatinal cervical abscess	Spontaneous; tenth day; opened into throat	Spontaneous with pus; bled from mouth and nose	Single	.....	.....	Death	Assumed that internal carotid artery was eroded
Lübbers: Ztschr. f. Ohrenh. **66**: 18, 1912	M 39	Recurrent angina 7 months; peritonsillar and parapharyngeal	Spontaneous	Spontaneous	Repeated small amounts for days; later two severe hemorrhages	External carotid artery	.....	Recovery	
Lübbers	F 40	Peritonsillar and parapharyngeal	Incision external; no pus; spontaneous into mouth 3 days later	Immediate with spontaneous rupture into mouth	Single	.....	.....	Death	Hole in plica pharyngo-epiglottica; foul exudate; erosion of superior laryngeal artery
Luke, cited by Newcomb and Chappell: Tr. Am. Laryng. A. 1908, p. 321	?	Peritonsillar	Spontaneous	Spontaneous	Recurrence 3 days later; also next day	Carotid artery	.....	Recovery	
Lukens, J. A.: Ohio State M. J. **25**: 879, 1929	F 16	Peritonsillar and parapharyngeal	Spontaneous	Spontaneous with abscess	Recurrent 2 and 4 days later; fatal hemorrhage next day	.....	.....	Death	False aneurysm of internal carotid artery, near base of skull; erosion into large abscess cavity

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Lukens	F 3	Peritonsillar and parapharyngeal	Spontaneous	Spontaneous	Recurrent 1 week, 15 and 16 days later; swelling of neck subsided after each hemorrhage	Common carotid artery	.....	Recovery	
Lyot, C., and Petit, R.: Gaz. méd. de Paris **10**: 1, 1897	F 19	One month after scarlet fever; pulsating mass below tip of mastoid pushing tonsil medially	Exploratory puncture; pure blood	.....	.....	.....	.....	Recovery	
MacGregor, cited by Lebram and Vergely: Ztschr. f. Ohrenh. **51**: 4, 1906	M 20	Postpharyngeal and parapharyngeal	.....	Spontaneous and fulminating; fatal from mouth and nose	.....	.....	.....	Death	Abscess at base of skull; erosion of internal carotid artery
Macmurdo: Chelius (South) **2**: 268, 1847	?	Suppuration of glands of neck	Spontaneous	Spontaneous	Recurrent	Common carotid artery	Bleeding stopped	Death 7 days later	Aneurysmal varix; destruction of bifurcation of carotid artery and internal jugular vein
Mahler: Internat. Centralbl. f. Laryng. **28**: 396, 1912	?	Peritonsillar	Incision	Followed day after incision	.....	.....	.....	Recovery	
Marotte: Bull. de Therapeut. **68**: 385, 1875	37	Peritonsillar	Spontaneous	Followed shortly after spontaneous opening of abscess	Recurrent	.....	.....	Recovery	
May: Ann. Surg. **1**: 559, 1885	F 13	Scarlet fever; swelling of parotid area up to mastoid	Spontaneous from ear	Spontaneous from ear 1 week later, ½ pint	Recurrent 2 days later, 1 pint; several smaller hemorrhages	Common carotid artery	Aphasia in 48 hours; sluggish movements of arms; recovered in 3 weeks	Recovery	
May	Child	Scarlet fever; abscess of neck	None	Spontaneous from ear	Plugging of ear followed by fatal hemorrhage from throat	Hoped to ligate but was too late	.....	Death	
Mayo, H.: London M. & Phys. J., December, 1828, p. 38; cited by Pilz: Arch. f. klin. Chir. **9**: 257, 1868	M 23	Syphilitic ulcer in pharynx	.....	Spontaneous	Recurrent	Common carotid artery	Fainting spells; vision impaired; recovered	Death 5 years later	Involvement of lingual artery

Mayo, R.: London M. & Phys. J., vol. 8; Froriep's Notiz. **3**: 80, 1837	M	Palate	Incision	Immediate on incision	Recurred next day	Carotid artery after first hemorrhage	.....	Recovery	
Mery: Bull. Soc. de chir. **2**: 282, 1870	..	Tonsil	.....	.....	.....	.....	.....	Death	Erosion of internal carotid artery
Messiter, M. A.: Lancet **1**: 61, 1883	M 24	Peritonsillar and parapharyngeal	?	Spontaneous fourth day	.....	Common carotid artery	.....	Recovery	
Metcalfe: New York M. Times **1**: 35, 1852	M 6	Scarlet fever; cervical	None	Spontaneous twelfth day	Recurred twice in short time	.....	.....	Death	Severity of bleeding probably due to internal carotid artery
Miegewille: Bull. Soc. de laryng. d. hôp. de Paris **10**: 11, 1926	.. 45	.....	Spontaneous	Spontaneous 2 days later	Recurrent every 2 days for 8 days	External carotid artery	.....	Recovery	
Miegewille	..	Peritonsillar	Incision	Twelve hours after incision; terrific	.....	Common carotid artery	.....	Recovery	
Mill: Edinburgh M. & S. J. **55**: 125, 1841	.. 3	Scarlet fever; submaxillary abscess	Incision	Sudden terrific hemorrhage from mouth and wound 6 days later	Single	.....	.....	Death	No autopsy; internal carotid artery probably eroded
Mill	F 7	Scarlet fever	.....	Sudden violent hemorrhage from throat eighteenth day	Single	.....	.....	Death	No autopsy; internal carotid artery probably eroded
Moizard: J. d. méd. et chir. prat. **57**: 347, 1886	M 41	Peritonsillar	Spontaneous	Next day from nose and mouth, more than 1 liter	Single	Prepared to ligate but deferred	.....	Recovery	Bleeding probably from branch of ascending pharyngeal artery
Monod, C.: Bull. et mém. Soc. d. chir. de Paris **8**: 666, 1882	..	Peritonsillar	Spontaneous eighth day	Spontaneous; fatal	Single	.....	.....	Death	Probably bleeding came from internal carotid artery
Moty: Zentralbl. f. chir. **34**: 143, 1907	..	Peritonsillar	.....	Spontaneous hematoma of wall of pharynx	Single	Common carotid artery	.....	Recovery	
Muller: Med. Cor.-Bl. d. württemb. ärztl. Landesver., 1885; cited by Newcomb: Tr. Am. Laryng. A., 1908, p. 321	M 44	?	Spontaneous	Spontaneous	Recurrent	.....	.....	Death	Facial artery thinned; ulcerated cavity full of pus at bottom of left tonsil
Murcheson: Tr. Path. Soc. London **10**: 99, 1859	M 8	Scarlet fever; abscess of angle of jaw	Incision on one side; bloody pus; spontaneous on opposite side	Spontaneous from opposite side	Recurred daily for several days	.....	.....	Death	Extensive cervical and parapharyngeal cellulitis; erosion of internal jugular vein

Peritonsillar, Retropharyngeal, Parapharyngeal and Cervical Abscesses with Hemorrhage—Continued

Author	Sex and Age of Patient	Infection	Evacuation: Spontaneous or by Incision	Hemorrhage; Spontaneous or Following Incision	Hemorrhage; Single or Recurrent	Ligation of Major Vessel	Disturbance Due to Ligation, If Any	Result	Autopsy Report and Comments
Nathan, D.: New York M. J. **100**: 24, 1914	M 21	Peritonsillar	Incision tenth day; tonsil necrotic and sloughed out in 7 days	Severe fatal hemorrhage next day; bleeding vessel seen in fossa; resisted clamps; tissue friable	Single	.....	.....	Death	
Negus: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **16**: 221, 1930	M 47	Peritonsillar	Incision	Hemorrhage on incision	Recurred 1 week later; tonsils removed; cured	.....	.....	Recovery	
Negus	M 13	Lateral part of pharynx	Incision	Spontaneous prior to incision	Recurrent; tracheotomy	External carotid artery seventeenth day	Hemorrhage followed	Death	Erosion of ascending pharyngeal artery into abscess cavity
Neuffer (1922), cited by Skoog: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**: 206, 1932	M 18	Pharynx (?)	?	Spontaneous twelfth day	Recurred next day	.....	.....	Death	Erosion of internal carotid artery; spurious aneurysm
Neumarkt: Internat. Centralbl. f. Laryng., Rhin. u. verw. Wissensch. **33**: 232, 1917	M Adult	Peritonsillar	Incision	Severe hemorrhage followed	.....	.....	.....	Recovery	
Newcomb: Tr. Am. Laryng. A., 1908, p. 321	M 55	Peritonsillar	Incision	Hemorrhage followed incision	Recurrence 36 hours later; packing; recurrence next day	Common carotid artery	Headache	Recovery	
Nicholls: St. George Hosp. Rep. **4**: 211, 1869	F 14	Scarlet fever; suppuration of glands of neck; erosion into throat; great slough	No	Spontaneous terrific hemorrhage; fatal	.....	.....	.....	Death	Erosion of internal carotid artery
Norton: The Throat and Larynx, London, Hardwicke, 1875, p. 12	F 4	Retropharyngeal	Incision refused	Spontaneous fatal hemorrhage	.....	.....	.....	Death	Erosion of internal carotid artery
d'Onofrio, F.: Arch. ital. di otol. **37**: 275, 1926	F 28	Peritonsillar	Not opened	Spontaneous through nose; swelling receded	Single	.....	.....	Recovery	
d'Onofrio	M	Peritonsillar	Not opened	Spontaneous through nose	Single	.....	.....	Recovery	
Ortmann, K. K.: Hospitalstid. **73**: 95, 1930	F 19	Parapharyngeal	Neither	Spontaneous from nose	Recurrent; cerebral embolism 1 week later	.....	.....	Death	Perforation of internal carotid artery; spurious aneurysm

Patterson, N.: Lancet **1**: 1256, 1926	F 31	Peritonsillar	Incision twice	Hemorrhage at second incision	Recurrence for 6 days	External carotid artery	.....	Recovery; long convalescence	
Pearlman: author's case	M 4	Retropharyngeal; parapharyngeal	Incision seventh day	Slight with incision	Recurred 48 hours later; fulminant; fatal	.....	.....	Death	
Pearlman: author's case	F 5	Retropharyngeal; parapharyngeal	Abscess not opened	Spontaneous seventh day; severe	Recurred after ligation	Common carotid artery	Miosis; bleeding recurred; carotid artery ligated again bleeding stopped	Recovery	
Pearlman: author's case	M 6	Retropharyngeal; parapharyngeal	Incision seventh day; no pus	Slight bleeding at incision	Severe next day	Common carotid artery; bleeding stopped	.....	Recovery	
Pepper, A. J.: Tr. Clin. Soc. London **16**: 21, 1883	M 30	Scarlet fever; edema of palate on left side; dyspnea	Incision after ligation	Spontaneous	Recurrent several times in one day	Common carotid artery	.....	Recovery	
Pircher: Wien. med. Wchnschr. **12**: 553, 1862	F 8	Angina; recidivation 6 days later; swelling of anterior pillar and trismus of palate	.....	Spontaneous, 5 ounces	Recurred several days later; fatal	.....	.....	Death	Rupture of false aneurysm of internal carotid artery above bifurcation
Pitts, 1882, cited by Lebram from Vergely: Ztschr. f. Ohrenh. **51**: 4, 1906	M 39	Peritonsillar 14 days	.....	Spontaneous	Recurrent	Carotid artery; fatal hemorrhage followed	.....	Death	Large hole in tonsil; inner surface of carotid artery covered by abscess; ulceration of carotid artery size of end of finger
Plewka: Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**: 129, 1931	? 1½	Fever 1 week; otitis media	.....	Spontaneous from ear	Recurrent 5 days later; fatal from nasopharynx	.....	.....	Death	Small abscess lateral to eustachian tube; rupture into auditory canal; two erosions into internal carotid artery
Porter, cited by Kennedy, H.: Some Accounts of Epidemic of Scarlatina, Dublin, Fannin & Co., 1843, p. 106	M 11	Scarlet fever; cervical abscess; necrosis of petrous bone	Spontaneous; pus from ears and cervical abscesses	Spontaneous from ear sixth week preceded by severe pain	Recurrent daily; severe; bright blood poured from ear; also from throat	.....	.....	Death by exsanguination	
Postempski, P.: Gazz. med. di Roma **13**: 73, 1887	M Adult	Throat	Spontaneous	Spontaneous 24 hours after abscess opened	.....	Common carotid artery	.....	Recovery	Internal carotid artery probably the seat of erosion
Ransohoff: Ann. Surg. **68**: 153, 1918	F 16	Angina; swelling of neck; incision, pus	Hemorrhage 5 days later from throat	Recurrent; pulsating mass lateral to pharynx; miosis of same side	Recurrent	Common carotid artery	.....	Recovery	Probable spurious aneurysm of internal carotid artery

Peritonsillar, Retropharyngeal, Parapharyngeal and Cervical Abscesses with Hemorrhage—Continued

Author	Sex and Age of Patient	Infection	Evacuation; Spontaneous or by Incision	Hemorrhage; Spontaneous or Following Incision	Hemorrhage; Single or Recurrent	Ligation of Major Vessel	Disturbance Due to Ligation, If Any	Result	Autopsy Report and Comments
Reeves, cited by Newcomb from Chappell: Tr. Am. Laryng. A., 1908, p. 321	?	Peritonsillar	Spontaneous	Hemorrhage same day; spontaneous	Recurrent from 6 to 8 times	Advised but declined	.....	Death from recurrent hemorrhage	
Reisman: Arch. f. Ohrenh. **125**: 309, 1930	?	Peritonsillar	Incised after spontaneous hemorrhage	Spontaneous	Recurrent day after incision; fatal	.....	.....	Death	Sudden death believed due to embolus
Rendu: Bull. Soc. anat. de Paris **14**: 66, 1839	M Adult	Abscess of throat, chronic	Hemorrhage followed attempt by patient to relieve self by passing knife blade into throat	Immediate copious hemorrhage	.....	.....	.....	Death	2 or 3 openings into postpharyngeal abscess; internal carotid artery intact; source of hemorrhage undetermined
Riecke, H. G.: Deutsche Ztschr. f. Chir. **205**: 347, 1927	F 4	Angina 10 days; swelling of angle of jaw; parapharyngeal	Neck opened outside after first hemorrhage	Spontaneous from right ear	Recurrent after incision	.....	.....	Death	Large parapharyngeal abscess; no erosion of large vessels; possible erosion of internal jugular vein at the bulb
Rivers: Bull. Johns Hopkins Hosp. **30**: 240, 1919	F 5½	Postscarlatinal slough of tonsil and palate; cervical	Cervical abscess incised	Hemorrhage 3 days later; marked exsanguination; transfusions	Severe recurrent erosion of external carotid artery and internal jugular vein	Common carotid artery ligated; jugular vein packed	Chvostek's sign; Babinski's sign; internal strabismus; loss of memory; loss of sense of pain; double clonus	Recovery	
Roberts, E. R.: Laryngoscope **40**: 807, 1930	Baby	Retropharyngeal; swelling of neck; pus and blood from ear	Incision	Spontaneous from ear	Recurrent when abscess of throat incised second time	External carotid artery and internal jugular vein; swelling of throat increased; aspiration followed by violent hemorrhage	.....	Death	
Robinson, F.: London M. Gaz. **10**: 376, 1850	M 42	Probably parapharyngeal	.....	Spontaneous	Recurrent	Common carotid artery	Rapid pulse; convulsions; cold body and limbs; normal fifth day	Death	Walls of carotid artery involved just below the bifurcation; erosion present
Rolleston: Tr. Am. Laryng. A. **38**: 124, 1913	F 22	Both tonsils; from measles	.....	Spontaneous following cough	Single; fatal	.....	.....	Death	Throat ulcerated; neither carotid artery involved; extensive streptococcic infection probably caused deep sloughing and erosion of small vessels

Roll-Hansen, D. H.: Med. Rev. **35**: 283, 1918	M 35	Phlegmonous angina	Incision	Four days after incision	Recurrent daily after fourth day following incision; severe	External carotid artery	.....	Recovery	
Ruttin: Ztschr. f. Ohrenh. **77**: 206, 1918	F 70	Peritonsillar following infection of ear	?	Spontaneous	Recurrent	.....	.....	Death	Infected hematoma in pharynx perforating below tubal opening; erosion of large vessel not proved
Sadowski, N.; Monatschr. f. Ohrenh. **66**: 731, 1932	..	Peritonsillar	Incision	Followed incision sixth day	Single; controlled by packing	.....	.....	Recovery	
Sadowski	..	Peritonsillar	Spontaneous	Continuous until tonsil sloughed out	.....	.....	.....	Recovery	
Salinger: author's case...	M 3½	Retropharyngeal	Spontaneous	Spontaneous	Recurrent, slight for 4 days; then two severe hemorrhages	Common carotid artery	Horner's syndrome; disappeared later; paralysis of hypoglossal nerve	Recovery	
Salinger and Pearlman (service of Jerome F. Strauss), Michael Reese Hospital	F 5	Retropharyngeal; parapharyngeal	Incision external fourteenth day	Bleeding 1 week after incision (from throat)	Continuous for several hours	.....	.....	Death while preparations being made for ligation	
Salinger and Pearlman (service of Harry Kahn), Michael Reese Hospital	M 10	Retropharyngeal; parapharyngeal	Incision seventh day; no pus	Severe 48 hours after incision	24 hours of intermittent bleeding; transfusion	.....	.....	Death	
Salinger and Pearlman (service of A. Levinson), Michael Reese Hospital	F 2	Retropharyngeal	Spontaneous thirteenth day	Bleeding when opening spread	Bleeding on next day; dyspnea	Superior thyroid; tracheotomy	Bleeding continued	Death next day	Cellulitis and fibrinous exudate; parapharyngeal abscess extending to pyriform sinus
Salinger and Pearlman (service of Jerome F. Strauss), Michael Reese Hospital	M 7	Retropharyngeal; parapharyngeal	.....	Spontaneous ninth day	Intermittent for several days	Common carotid	Bleeding continued; jugular vein ligated	Death	Cellulitis of neck; false aneurysm of common carotid artery; rupture into pharynx
Savory: Brit. M. J. **2**: 706, 1880	M	Neck	Spontaneous	Spontaneous through external opening 3 days later	Single	.....	.....	Death	Common carotid artery ruptured
Schlemmer: Arch. f. Laryng. **33**: 412, 1920	.. 38	Necrotic angina; plastic edema of neck; edema of larynx; tracheotomy	Collar mediastinotomy	Spontaneous from throat sixth day	Recurrent	External carotid artery	Metastatic pneumonia	Recovery after 5 weeks	Hemorrhage probably came from ascending pharyngeal artery; ulceration deep in tonsillar region
Schmiegelow: Zentralbl. f. Laryng. **17**: 594, 1901	M 30	Peritonsillar	Not stated	Spontaneous fourth day	Single	.....	.....	Recovery by palliative measures	

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Scholz, W.: Wien. med. Presse **6**: 595, 1865	?	Diphtheritic angina	.....	Spontaneous	.....	.....	.....	Death	Erosion of internal carotid artery; thrombosis of internal jugular vein
Sedgwick: London M. Gaz. **13**: 581, 1851	M 4½	Scarlet fever; cervical	Incision fifteenth day; pus and blood	With incision	Recurred same evening; mass increased; free incision; clots removed; terrific bleeding	.....	.....	Death	Large erosion of internal jugular vein
Sercer, A.: Monatschr. f. Ohrenh. **62**: 648, 1928	F 22	Peritonsillar about 6 weeks after onset of sore throat	Incision	Hemorrhage 15 days after incision; from erosion in pharynx farther back	Recurrent	External carotid artery first; continued bleeding; common carotid artery ligated, false aneurysm present	Complete hemiplegia with aphasia third day	Death 4 weeks later	Erosion of internal carotid artery; abscess of brain
Simeoni, C.: Arch. ital. di otol. **38**: 153, 1927	M 28	Peritonsillar; parapharyngeal; extensive	Incision; small amount of pus and some blood	Small amount at incision; 6 hours later severe hemorrhage, 2 liters; swelling not reduced	Recurrent next day; fatal	Considered but not performed	.....	Death	
Skoog, T.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**: 206, 1932	.. 7	Mumps 2 weeks; swelling of jaw; swelling of tonsillar area	Incision of angle of jaw; no pus; tonsillectomy; tonsil in phlegmonous base; vessels bare	Spontaneous from ear; fatal hemorrhage from throat after tonsillectomy	.....	.....	.....	Death	Erosion of internal carotid artery near cranial base; false aneurysm eroding auditory canal
Skoog	F 41	Peritonsillar	.....	Spontaneous profuse, 0.5 liter; small hole near upper pole; large hematoma	Recurrent, slight oozing	.....	.....	Recovery	Probably from branch of external carotid artery
Skoog	M 19	Peritonsillar	Incision	Hemorrhage several days after incision	Single	External carotid artery; loop thrown about common carotid artery	.....	Recovery	Compression of external carotid artery stopped bleeding; therefore, common carotid artery not ligated

Smith, R. W.: Dublin Quart. J. M. Sc. **1**: 505, 1846	M 9	Scarlet fever; cellulitis of neck	Incision of neck tenth day; foul pus	Hemorrhage fifteenth day	Recurred 4 days later	.....	.....	Death	Sloughing in neck; circular erosion of internal jugular vein; tonsil sloughed
Sokoloff, A.: Monatschr. f. Ohrenh. **45**: 333, 1911	M 2	Retro-pharyngeal	Incision	Immediate on incision	Single	.....	.....	Death	Erosion of internal carotid artery lying in abscess extending to base of brain
Spence, J.: London & Edinburgh Monthly J. M. Sc. **14**: 178, 1852	F	Throat; para-pharyngeal; tonsil destroyed	Incision 1 day after spontaneous bleeding	Spontaneous	Recurrent 2 hours after incision, continued for 24 hours	.....	.....	Death	Erosion of both internal and common carotid arteries lying in abscess cavity
Stoupy, E.: Arch. de méd. et pharm. mil. **28**: 177, 1896	M 22	Tonsils and glands enlarged	Spontaneous	Began 8 days after abscess opened; spontaneous from mouth and nose	Recurrent for 2 days; neck incised; clots	External carotid artery; bleeding continued	.....	Recovery	
Stumpf: Ztschr. f. Laryng. **9**: 385, 1920	M 13	Peritonsillar	Incision; no pus	Blood on incision; incision repeated; bleeding; tampon	Recurrent in small amounts for few days; clots removed; next night severe hemorrhage; fatal	.....	.....	Death	Extensive abscess to base of skull, 2.5 cm. square; erosion and aneurysmal dilatation of internal carotid artery
Stumpf	F 5	Sore throat 2 weeks; swelling of neck; peritonsillar swelling	Incision after ligation of carotid	Bleeding from ear several days prior to incision of abscess	Recurrent	Common carotid artery	Miosis; bleeding 4 days later; wound reopened; another ligature applied	Death	Adventitia separated from media to innominate vein; external carotid artery empty; internal carotid artery contained clot above erosion into abscess cavity
Syme, J.: London & Edinburgh Month. J. M. Sc. **2**: 961, 1842	M 9	Sore throat; swelling of neck for 5 months	Five months after onset abscess ruptured into auditory canal	Bleeding day after opening of abscess	Recurrent next day from throat; also several days later; numerous	Common carotid artery	Slight bleed from mouth and nose	Recovery	
Szekeres: Orvosi hetil. **24**: 1075, 1880	?	Retropharyngeal; post-scarlatinal	.....	Spontaneous	.....	.....	.....	Death	Perforation of common carotid artery; simultaneous erosion of esophagus
Tacey: Glasgow M. J. **1**: 405, 1868	M 25	Retropharyngeal	Spontaneous external	Bleeding 3 days after abscess ruptured	Recurrent next day; fatal	.....	.....	Death	Erosion of internal carotid artery near base of skull
Tawse, H. B.: J. Laryng. & Otol. **44**: 257, 1929	M 49	Peritonsillar	Incised after ligature, 3 weeks from onset	Spontaneous fifteenth day	Recurrent for 2 days; severe one week later	External carotid artery; provisional loop about common carotid artery; removed in 10 days	.....	Recovery	

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Terganowan: Tr. New Jersey M. Soc., 1874, p. 195	M 20	Sore throat; peritonsillar and parapharyngeal	Spontaneous 3 months after onset of illness	Spontaneous	Single	.....	.....	Death	Complete destruction of parotid gland; gangrenous softening of entire circumference of external carotid artery with fistulous opening into throat
Thomasson, W. J.: Two Cases of Hemorrhage Following Peritonsillar Abscess; J. A. M. A. **76**: 931 (April 2) 1921	M 45	Peritonsillar	Incision	Hemorrhage 10 days after incision, 1 pint	Continued oozing; recurrent next day; severe; packing	.....	.....	Recovery	
Thomasson	M 48	Peritonsillar, bilateral	Spontaneous	Spontaneous with pus	Continued oozing; packing; bleeding stopped in 24 hours	.....	.....	Recovery	
Thornval, cited by Skoog	M 19	Peritonsillar	Incision	Immediate on incision	Single	Common carotid artery	.....	Recovery	
Tollet, W.: Finska. läk.-sällsk. handl. **72**: 305, 1930	.. 11 mos.	Swelling angle of jaw; parapharyngeal	.....	Spontaneous from ear	Single	.....	.....	Death	Erosion of internal carotid artery; spurious aneurysm
Tollet	M 18	Tonsil	Incision	Pulsating swelling above tonsil 6 weeks; later puncture; blood	Only on needle puncture	Common carotid artery	.....	Recovery	
Tondorff: Internat. Zentralbl. f. Ohrenh. **32**: 353, 1930	M 6	Peritonsillar and parapharyngeal	Incision; no pus	Spontaneous from ear; also from incision; mastoid opened; normal	Fatal hemorrhage before ligation could be done	.....	.....	Death; hemiplegia appeared shortly before (12 hours)	
Travers, F. T.: Brit. M. J. **2**: 703, 1902	M 24	Peritonsillar	Incision	Bleeding 4 days later	Recurrent	Internal jugular vein; bleeding recurred; ligation of common carotid artery	.....	Recovery	
Trutnev: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **8**: 292, 1925	..	Peritonsillar and parapharyngeal 2 weeks	.....	Spontaneous, 1½ glasses	Recurrent 5 days later; fatal	.....	.....	Death	Erosion of internal carotid artery close to bifurcation; abscess cavity below tonsil
Tweedie: Proc. Roy. Soc. Med. (Sect. Laryng.) **6**: 15, 1912-1913	M 1 yr. and 8 mos.	Peritonsillar 3 weeks; cervical adenitis	Incision	Spontaneous from ear; from throat following incision	Continuous	Incision for ligation; death before operation completed	.....	Death	Erosion of internal carotid artery near base of skull

				incision		completed			
Valence: Arch. de méd. nav. **100**: 279, 1913	M 21	Parapharyngeal; submaxillary from infection of tooth	Incision submaxillary; foul pus	Four days following incision; from wound	Recurrent	Common carotid artery	Choreiform movement of arms, legs next day; athetosis; lessened in a few days	Recovery	
Veillard: Arch. internat. de laryng, cited by Newcomb: Tr. Am. Laryng. A., 1908, p. 321	M 26	Peritonsillar	Spontaneous	Spontaneous	Recurring	.....	.....	Recovery	
Vergely: J. de méd. de Bourdeaux **15**: 545 and 559, 1886; cited by Newcomb	M 30	Peritonsillar; submaxillary; dyspnea	.....	Spontaneous	Single	.....	.....	Death	Rupture of internal carotid artery 2 cm. above bifurcation
Vergely	?	Peritonsillar	.....	Spontaneous	Single	.....	.....	Death	
Volmat: Rev. de laryng. **47**: 5, 1926	? 29	Peritonsillar	Incision	Bleeding 10 days after incision	Single	External carotid artery; bleeding stopped	.....	Death 48 hours after ligation; septicemia	
Walker, 1825, cited by Newcomb from Chappell: Tr. Am. Laryng. A., 1900, p. 213	..	Bilateral peritonsillar; swelling in front of thyroid	Incision of external abscess; communicated with peritonsillar abscess	Two days after incision, from mouth and nose; terrific	Single	.....	.....	Death	
Walliczek: Folia otolaryng. **34**: 374, 1931	F 26	Peritonsillar; recurrent	Incision repeated 4, 13 and 20 days later	Followed last incision	Single	.....	.....	Death while on table prepared for ligation	Erosion and aneurysm of internal (?) maxillary artery
Watson, cited by Newcomb from Chappell: Tr. Am. Laryng. A., 1900, p. 213	..	Peritonsillar	Incision	Immediate on incision	Recurred same day and next day; recurred 2 weeks later; fatal	.....	.....	Death	Erosion of lingual artery
Weinlechner: Wien. med. Bl. **8**: 1624, 1885	M 37	Peritonsillar	Spontaneous	Spontaneous 10 days after incision	Recurrent	Common carotid artery	Hemiplegia; delirium; complete recovery in 6 weeks	Recovery (patient died 10 months later of apoplexy)	
Wishart, D. E. S.: Canad. M. A. J. **13**: 635, 1924	? 18 mos.	Sore throat 3 weeks; swelling 2 weeks; retropharyngeal and parapharyngeal	Spontaneous	Spontaneous	Continuous	.....	.....	Death	Erosion of internal carotid artery
Wulff, P.: München. med. Wchnschr. **47**: 687, 1900	F 9	Peritonsillar; later parapharyngeal	Attempt to incise; gagging; 3 weeks later swelling and spontaneous rupture of abscess on opposite side	Bleeding from attempt to incise; later pulsating swelling in pharynx	Recurrent on puncture of swelling	Common carotid artery	Aneurysmal sac suppurated and ruptured into auditory canal	Recovery	
Wylie, A., and Wingrave, U. H. W.: Ztschr. f. Laryng. **9**: 385, 1920	F Tuberculous case	Retropharyngeal	Incision	Fatal hemorrhage 9 days after incision	.....	.....	.....	Death	Erosion of internal carotid artery

As for such minor complications as enophthalmos and miosis, it was frequently found that these antedated ligation of the carotid artery, and were undoubtedly due to involvement of the carotid artery and the upper cervical sympathetic nerve in the suppurative process. Paresis of the hypoglossal and facial nerves and involvement of the glossopharyngeal and spinal accessory nerves, which occurred in several cases, are easily explained as jugular fossa syndromes the result of cellulitis and suppuration at the base of the skull. Fortunately in the cases in which recovery occurred, most of these manifestations disappeared in time, so that one is quite safe in making a favorable prognosis when such symptoms supervene.

On summing up this phase of ligation of the common carotid artery, one finds on the basis of the accumulated experience of a century that 25 per cent of all ligations of the common carotid artery, regardless of age or ailment, are accompanied by serious cranial complications of which at least one-half are fatal. One can then truthfully say that the deaths directly caused by ligation should not number more than 10 or 15 per cent of the total, because, as has been previously pointed out, there are other causes contributing to the generally accepted higher mortality, such as sepsis, shock and acute anemia. Therefore, one should not magnify the dangers of ligation of the common carotid artery, but when confronted with a serious hemorrhage or recurring hemorrhages should rather consider the dangers and high mortality that may be expected from nonintervention and accept ligation of the common carotid artery as much the lesser of two evils. It, at least, offers a two to one chance for recovery as against almost certain death (table 1).

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BRANCHIAL FISTULA

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This report is made because of the infrequent occurrence of the complete branchiogenic fistula with the external and internal openings present and without cystic enlargement throughout the fistulous tract. The case which I am reporting has the distinctive characteristics of the complete branchiogenic fistula. Six patients with this condition were operated on in the Massachusetts General Hospital and in the Massachusetts Eye and Ear Infirmary during the years 1926 to 1931.

According to Thompson,[1] a satisfactory classification of cysts of the neck of branchial origin is:

1. Lymphatic cysts. Located in the lower part of the neck in the posterior triangle. They are derived from the jugular sinus and may extend under the clavicle.

2. Thyroglossal cysts, which occur in the midline as lingual cysts, or below the hyoid under the deep fascia. They are derived from the thyroglossal duct which is formed by a bud of hypoblast which grows down between the mandibular and the pharyngeal parts of the tongue and eventually forms the isthmus of the thyroid.

3. Dermoid cysts found in the midline and formed during the fusion of the ventral folds.

4. Branchiogenetic cysts. Derived from persistent remains of the external branchial depressions of the embryo.

In order to understand the source of cysts of branchial origin it is necessary to know the fate of the arches and clefts in the human embryo. The arches are visible in the third week and are separated by depressions, in the fish by clefts. The depressions are separated by a membrane which is covered on its outer aspect by epiblast and on its inner side by hypoblast. In the normal course of events this membrane disappears. In the rare instances in which it is perforated a complete cleft results, which may persist in the adult as a branchial fistula. By the sixth week all evidence of the branchial arches and depressions has vanished.

Of the branchial clefts or depressions four can be recognized in the embryo. The first, the hyomandibular, persists. Its external depression is represented by the external auditory meatus, and in connection with it the eustachian tube and the tympanum develop. The membrana tympani represents the cleft membrane.

From the Clinical Conference of the New England Hospital for Women and Children, Roxbury, Mass., the Massachusetts Eye and Ear Infirmary and the Massachusetts General Hospital, Boston.

1. Thompson, J. E.: Surgery and Embryology, Surg., Gynec. & Obst. **31**:18, 1920.

The second, third and fourth clefts disappear by the downward growth of the second arch forming the cervical sinus. Normally this should disappear also, but if it persists a cyst is formed. The cyst may open externally on the neck along the anterior border of the sternomastoid muscle. Occasionally the cyst opens into the pharynx. When this occurs the opening into the pharynx usually passes through the membrane of the second cleft. When both internal and external openings are present, a long fistulous tract lined with epithelium stretches between the surface of the neck and the cavity of the pharynx. This is called the branchiogenetic fistula. If the tract is dissected out it always passes above the fork of the carotids, i. e., above the third branchial arch. The internal opening is in the tonsillar recess (second cleft depression). The external opening varies in position, but is always along the sternomastoid muscle.

Roentgenogram of branchiogenic fistula on the right side of the neck, extending upward from ½ inch (1.5 cm.) above the clavicle into and through the right tonsil.

REPORT OF A CASE

J. P., aged 7 years, was admitted to the New England Hospital for Women and Children, Roxbury, Mass., on March 19, 1931, because of a discharging sinus in the neck. There had been frequent colds in the head, attacks of fever and persistent dry cough. There was a small, indurated mass ½ inch (1.3 cm.) square on the right side of the neck at the edge of the sternocleidomastoid muscle, about 1 inch (2.5 cm.) above the clavicle. There was a sinus in the center of this thickening, which showed a small amount of serous discharge. The mass was just under the skin and could be felt in the neck as a thick cord running up as far as the hyoid bone, where it was lost. For the purpose of diagnosis, iodized poppy seed oil 40 per cent was injected into the sinus and a roentgenogram was taken. By means of x-rays the sinus was shown passing into the throat. Methylthionine

chloride, U. S. P., (methylene blue) was then injected into the sinus, and this could be seen coming out into the throat through the tonsil at about its center.

Two days later a condition that appeared to be acute tonsillitis developed, with a temperature of 103.2 F. There was a membranous patch, limited to the right tonsil, which was apparently a local reaction to the injections of the dye and iodized oil into the fistula. The external opening formed an abscess and necessitated an incision for drainage.

Examination of the blood gave normal results, and the cultures from the throat showed streptococci and staphylococci.

The tonsils and adenoids were removed under general anesthesia on May 5. The tonsils were enucleated without difficulty. The right tonsillar fossa appeared clean and was carefully explored for the exit of the fistulous opening, but none could be demonstrated. The dye was not used at this time. The right fossa (on the side of the fistula) had a persistent granulation in the center, but after the operation on the fistula the tonsillar fossa became smooth.

Microscopic examination of the tonsil showed a central sinus lined with squamous epithelium.

The operation on the branchial fistula was performed on May 26, 1931, at the Massachusetts Eye and Ear Infirmary by Dr. Frederick Garland and myself. Ether anesthesia was used. Methylthionine chloride was injected into the fistula, which was observed to come from the middle of the tonsillar fossa. A probe was inserted into the fistula. The incision was made through the skin from about 1 inch below the mastoid to 1 inch above the clavicle. It passed along the line of the probe encircling the external opening. By blunt dissection and use of the scissors, the fistulous tract which lay just below the fascia was freed easily as high as the level of the hyoid bone, where it dipped deep into the tissues of the neck. There were no branches or cystic enlargements. The common carotid lay posterior and external to the fistular tract. The end of the probe could be felt in the tonsillar fossa with the finger introduced into the throat. The tract was readily freed up to the tissues of the throat, but the throat was not opened. Ligatures were placed as close to the throat as possible, and the fistula was cut off. Cutaneous sutures were placed. The patient was discharged in five days.

Microscopic examination showed a sinus tract lined by stratified squamous epithelium.

The scar resulting from the incision in the neck healed with no disfiguring contraction. The cough gradually disappeared. Hyndman and Light [2] mentioned the occurrence of a dry cough due to the proximity of the vagus nerve.

On June 29 (five weeks after the operation on the fistula) the patient was admitted to the ward at the New England Hospital for Women and Children with pneumonia of the lower lobe of the right lung. The onset was abrupt, with chills and vomiting. On admission the temperature was 104 F., the pulse rate 130 and respiration 35. A roentgenogram of the chest showed the area of involvement. The patient made an uninterrupted recovery following typical pneumonia. A year later he had a second attack of pneumonia.

Hyndman and Light,[2] in a most complete study of branchial cysts and fistula, reviewed sixty-two case reports collected from various pub-

2. Hyndman, O. R., and Light, G.: Branchial Apparatus: Its Embryologic Origin and Pathologic Changes to Which It Gives Rise, with Presentation of Familial Group of Fistulas, Arch. Surg. **19**:410 (Sept.) 1929; J. Iowa M. Soc. **20**:260 (June) 1930.

lications up to 1929. No differentiation was made in their study between cases of cysts and those of a complete fistulous tract with an external and internal opening. A study of their table reveals that in a small proportion of the patients there were complete tracts.

In a study of the records of the Massachusetts Eye and Ear Infirmary and the Massachusetts General Hospital I found that during the past five years about sixty patients have been operated on for cysts of the neck of branchiogenic origin, but that only six of these had a definite fistulous tract. A brief outline of these six cases is given here.

Case 1.—E. S., aged 11 years, was admitted to the hospital on April 29, 1930. This patient had an aperture the size of a pin-point to the right of the trachea and a little below the thyroid, from which there exuded occasionally a small amount of thin, clear fluid. This had been present since birth. For several years she had received both roentgen treatment and injections of some caustic medicine into the sinus. The patient had always had a cough, because of which she had been kept in a sanatorium for months at a time, but a diagnosis of tuberculosis had never been made, and no improvement in the condition of the fistula had ever been noted. The tonsils and adenoids were removed two years previously. At operation, an oblique incision was made on the right side of the neck which enclosed the opening of the fistulous tract. This admitted a probe that could be passed into the pharynx in the region of the tonsil. The tract was dissected almost up to the level of the tonsil, where it was ligated and cauterized. No pathologic report was filed with the history.

This patient had a definite fistulous tract stretching between an external and an internal opening and an external sinus which had been discharging intermittently since birth. In view of the fact that she had been subjected for five years to constant treatment in various institutions and at home, with no improvement, the conclusion readily arrived at is that an early operation on the fistulous tract would be the method of choice.

Hyndman and Light stated that the injection of corrosive substances has given unsuccessful results; the preferred treatment is complete removal by a competent surgeon.

Wangensteen [3] placed emphasis on the necessity of a differential diagnosis of branchial cyst from tuberculous adenitis. To avoid long, unnecessary treatment, he found roentgen examination valuable. In the branchial cyst, the opaque medium gives a sharply circumscribed outline, he stated, while in a tuberculous lymph node or cold abscess formation, the opaque medium shows an irregular outline. The lipoid content imparts a shimmer or yellowish sheen to the fluid.

Case 2.—A. Z., aged 15, had a history of a chronic discharging sinus in the right side of the neck since the age of 5 years. There had been an intermittent discharge from the external opening, which was located 2 inches (5 cm.) above the clavicle. Tonsillectomy and adenoidectomy had been performed when the patient was 5 years of age. At operation, a vertical incision was made along the sternocleidomastoid muscle, and the sinus tract was dissected out. It passed in front of

3. Wangensteen, O. H.: Differentiation of Branchial from Other Cervical Cysts by X-Ray Examination, Ann. Surg. **93**:790 (March) 1931.

and anterior to the carotid sheath. A probe was passed through the sinus and out through the upper pole of the tonsillar fossa. The lower pole of the fistula was cut away, and the upper part was tied onto the probe, which was pulled through into the mouth. The sinus was fixed by suture at the point where it emerged at the upper pole of the tonsillar fossa.

Pagenstecher,[4] in his article on the classification of cysts of branchiogenic fistulas and their treatment, advocated complete removal, with inversion of the tract into the mouth and excision of the internal orifice with the attached tract.

CASE 3.—M. J., aged 36, who was admitted to the hospital on May 6, 1928, gave a history of a periodically draining sinus located 1 inch above the clavicle, on the border of the sternocleidomastoid muscle. It had been present since the patient was 4 months old. At operation, a transverse incision 1 inch in length was made just above the fistulous opening, which was followed up in front of the carotid sheath. A second oblique incision was made below the angle of the jaw. A well defined tract was followed to the level of the tip of the styloid process and appeared to enter the pharynx, but its connection could not be made out; the fistula separated and pulled off at this point and could not be picked up again. It passed anterior to the internal carotid. The patient was discharged one week after the operation.

Microscopic examination showed a filamentous cord 6 cm. long, lined with columnar squamous epithelium.

CASE 4.—A. D., aged 13, admitted to the hospital on Aug. 22, 1927, gave a history of having had an intermittent, discharging sinus on the left side of the neck since 3 months of age. The discharge consisted of a thin, yellowish material. Tonsillectomy and adenoidectomy had been performed five years previously. At operation, a curved horizontal incision around the external opening was made. There was a small fistula extending up to the deep tissues of the neck, which was lined with squamous epithelium.

CASE 5.—N. F., aged 7 years, admitted to the hospital on April 23, 1929, gave a history of having a small fistulous opening in the right side of the neck since birth. A thick cord could be felt extending from 1 inch above the clavicle at the border of the sternocleidomastoid muscle to the angle of the jaw. At operation, an incision was made through the skin and fascia of the right side of the neck, and the fistulous tract was dissected out. It showed a squamous epithelium lining.

CASE 6.—C. T., admitted to the hospital on June 26, 1931, gave a history of having had a discharging sinus in the neck, which followed the extraction of some teeth two years previously. The history did not state whether a fistulous opening had been observed before the extraction of the teeth. Tonsillectomy and adenoidectomy had been performed. At operation, an elliptic incision was made from behind the angle of the jaw and encircling a small external sinus located 1 inch above the clavicle. There was a definite sinus tract, which was followed deeply into the neck under the epigastric muscle to the pharyngeal wall. There it was tied and cauterized. Microscopic examination showed no remains of an epithelium lining.

SUMMARY

The first three patients had well defined fistulous tracts that allowed the passing of a probe from the external opening to, or into, the throat.

4. Pagenstecher, G. A.: Congenital Abnormalities of the Neck, Texas State J. Med. **25**:786 (April) 1930.

The external opening had been observed since birth. This classifies them as complete fistular tracts of branchiogenic origin. The fourth, fifth and sixth patients had fistulous tracts, but these were not as successfully demonstrated as in the first three cases.

These six cases of fistula were found scattered among the sixty cases classified in the files as cysts of branchial origin. The histories of all but the six patients whose cases have been reported showed no fistulous tract, and the external discharging sinus, in most instances, had been of less than one year's duration.

CONCLUSION

The true fistula is a complete tract which passes from an external opening just above the clavicle along the border of the sternocleidomastoid muscle and opens into the throat. It is present in the neck from birth. Microscopic examination shows this fistulous tract to be lined with squamous epithelium. These distinctive diagnostic points are all found in the case reported here.

PRINCIPLES UNDERLYING CILIARY ACTIVITY IN THE RESPIRATORY TRACT

I. A METHOD FOR DIRECT OBSERVATION OF CILIA IN SITU AND ITS APPLICATION

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It is in keeping with the generally increasing conservatism in the treatment of sinusitis and other nasal disorders that investigations of the fundamental principles applicable to the ciliated and secretory elements of the mucous membrane lining the respiratory tract should be made available. It is important to know what the primary effect of many reagents on the cellular elements of the membrane is, the rôle played by the nervous system in regulating ciliary activity, the adaptability of injured and regenerated tissue to changes in the environment and many other equally significant cytophysiologic relationships of this tissue. This series of publications, based on investigations of the past several years and the new problems resulting from earlier studies dealing with the phases of the subject already mentioned, is presented primarily to meet the needs of the rhinologist.

Numerous methods have been employed in the last hundred years to evaluate ciliary activity. A classification and brief description of the principles involved in them have already been published.[1] A method was described at that time in which the movements of cilia arranged in rows could be analyzed photographically with considerable exactitude. Its chief limitation was the necessity of using transmitted light, which factor required either that the animals studied be very small and transparent or else that the tissue be removed from the body.

The following method is applicable to cilia arranged on a smooth or on an irregular surface, the condition in which they usually occur and in which they always exist in the mammalian respiratory tract. Reflected transmitted light is used and the cilia may be examined in situ.

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1. Lucas, A. M.: Coordination of Ciliary Movement: I. Methods of Study and the Relation of Ciliary Coordination to Ciliary Inhibition, J. Morphol. **53**:243 (June 5) 1932.

The apparatus used is shown in figure 1. The principal requirements are that the light be fairly strong and concentrated, that the heat be removed, that the objective lens have a long working distance and that the microscope and the platform holding the preparations be easily and quickly adjustable to any angle which still permits light to be reflected from the ciliated surface through the lens system.

As pointed out previously,[2] the microscope is focused on a "highlight," and, in general, the greater the angle between incident and reflected beams, the greater is the visibility of the ciliary waves. The

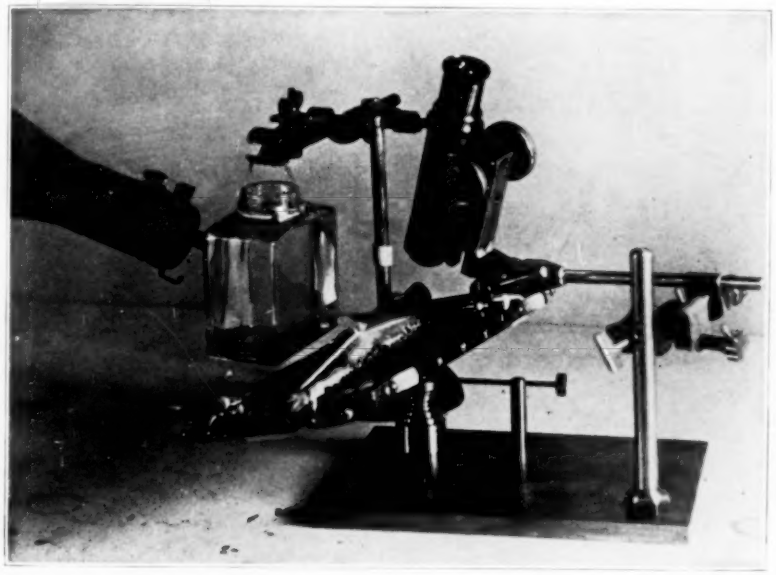

Fig. 1.—The apparatus designed to employ reflected light for direct observation of ciliary movements in situ. The microscope in focus on the roof of the mouth indicates the long working distance available.

principal advantage of this method is the fact that the movement of the cilia is visible at low magnifications; in fact, it has been observed with the naked eye, and quite readily with the aid of a watch-maker's lens, and the direction of this movement is discernible under the dissecting microscope with a magnification of 30 diameters. Higher magnification required for closer study is best obtained by increasing the power of the ocular. The combination of lenses used for these investigations has been a Spencer 5.4 × objective and a 20 × ocular, giving

2. Lucas, A. M.: Comparison of Ciliary Activity Under in Vitro and in Vivo Conditions, Proc. Soc. Exper. Biol. & Med. **30**:501 (Jan.) 1933.

a total magnification of 78 diameters. The advantage of the long working distance is threefold. First, the front lens and its metal holder are not in conflict with the turned-up edges of the tissue structures supporting the ciliated surface, as for example in the sinuses, nasal cavity and frog's pharynx. Second, great depth of focus, which is, in general, a corollary of a long working distance, permits a considerable portion of a sloping field to be clearly defined. The necessity of working with a field set at an acute angle to the optical axis will be discussed later. Third, the size of the visible field is large. With this particular lens its diameter is about 3 mm.

Two mechanical improvements not indicated in figure 1 are desirable. The arm to the microscope tube should be attached somewhere near the middle instead of at the bottom of the bracket, because in the photographed location it sometimes conflicts with the tilting of the platform. Secondly, an advantageous replacement is that of more rigid vertical and horizontal arms as well as a universal joint to eliminate vibrations. The arrangement shown in figure 1 is not sufficiently steady for photomicrography.

The platform holding the specimen is easily adjusted by means of the ball and socket pivot, so that the light may be caused to be reflected as a "high-light" from any portion of the ciliated surface desired for study. The microscope is then adjusted to coincide with the axis of the light reflected to the unaided eye.

If it is desired to keep the preparation in a moist chamber, the annoying reflection from the glass cover may be eliminated by placing in front of the heat filter a screen having a horizontal slit about 5 mm. wide. It can be adjusted so that the light passing to the glass in the line of vision is eliminated, but is reflected in the usual way from the ciliated surface lying a short distance below.

The factors which make it possible to see the ciliary waves at a magnification much less than is required to see them equally well by transmitted light are diagramed in figure 2. The mucous layer is not shown, but it is a thin sheet following the contour of the ciliary formation. It may be disregarded in the present description. The beam of light strikes the ciliated surface at an acute angle. When the cilia are at rest, all the light is reflected from the relatively smooth surface as shown at the right of figure 2 (*Q. C.*). It might be mentioned that the cilia at rest maintain an inclined position. When the cilia become active they beat metachronously and appear as low protuberances (*V. C.*) which move a short distance and disappear. These events have been described in diagrams by Proetz.[3] The light striking the crest of the

3. Proetz, A. W.: Five Preliminary Notes on Nasal Function, Ann. Otol., Rhin. & Laryng. **41**:1117 (Dec.) 1932.

wave is reflected from it at the same angle as the rays from the quiescent ciliated surface, and is visible through the microscope as a bright spot against a more or less dark ring. The contrasting dark areas are produced by two factors. One is the shadows cast owing to the interruption of light, and the other is the reflection of light from surfaces not parallel to the plane *A A'*. These reflected rays are indicated by dotted lines, and since they do not reach the microscope their points of origin appear black.

As is evident, there are certain points of similarity to the visibility of particles in dark-field illumination, but there are also differences, in that the light comes from only one direction and especially in that the background is bright instead of dark.

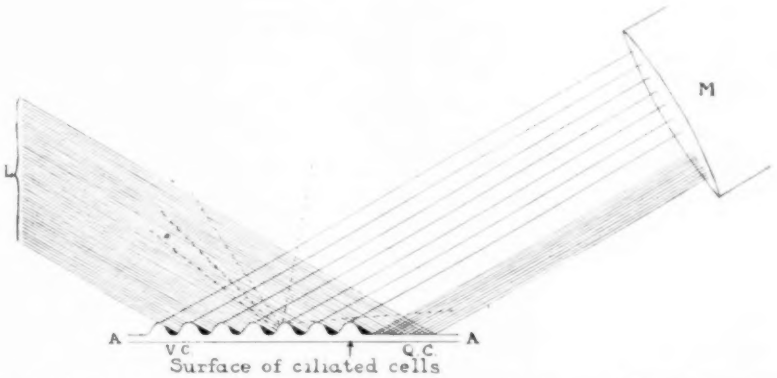

Fig. 2.—A diagram of the physical factors involved in rendering the ciliary waves visible by reflected light. *Q.C.*, quiescent cilia; *V.C.*, vibrating cilia; *L.* light from the lamp; *M*, microscopic objective.

Theoretically it should be possible to shift the microscope to some position other than the one receiving reflected light from the surface of the epithelium and still obtain beams reflected from the waves as indicated by the dotted lines. It is apparent that the bright background would be lacking. Such a condition has been observed, but it is not suitable for study of the waves because only a point of light is visible and the contour of the waves is lost.

The angle between incident and reflected rays may be varied if the same angle in relation to the normal axis is maintained; for most work a total angle of about 90 degrees is desirable. If the angle is greatly decreased beyond this, the light reflected from the bottom of the waves added to that from the crests renders the correspondingly decreased dark points between them invisible to the eye.

It has been found in working with the cilia of the mammalian nose that the movements of the ciliary waves can be better studied when the waves are moving toward the observer than toward the source of light, which difference is probably caused by the form of the wave.

The reflection method makes possible the direct observation and record of ciliary activity and eliminates the necessity of applying particles of carbon and other substances to the surface. Moreover, it has been shown [2] that particles of inert materials applied to the mucous film covering the cilia in the frog are stimuli which activate the normally quiescent cilia into a state of energetic beating. This method has been applied also to studies on the relationship between mucous progression and ciliary activity and to an investigation of nervous stimulation of cilia, work which will be reported soon. It has been used likewise to correlate the direction of mucous flow with the direction of ciliary beat, especially after injury to the epithelium.

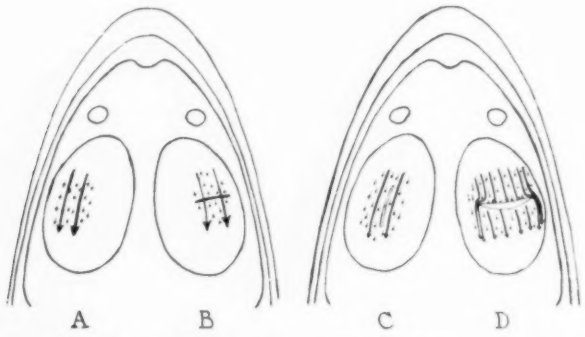

Fig. 3.—The direction of ciliary and mucous movements in the roof of the frog's mouth. The small arrows designate the ciliary movement and the larger ones, the mucous flow.

Minor injuries to the ciliated mucous membrane of the human nose are frequently adjuncts of the treatment required. It is important to know whether the ciliated cells surrounding the point of injury are sufficiently adaptable to change their polarity to meet the new conditions which result and hence to facilitate the passage of overlying particles around the injured area. Frogs were used for this purpose because, owing to their lower position in the series of phylogenetic relationships, they might more readily reveal this tendency than animals of a higher order. The roof of the mouth is shown in figure 3, and the four conditions of the experiment are designated *A* to *D*. *A* shows the normal uninjured condition. The large arrows indicate the direction of mucous flow; the smaller ones, the direction of the effective beat of the cilia. *B* shows the result of a single cut across the paths of ciliary and mucous

movement. Neither is affected; the cilia beat in the same direction as before, and the mucus because of its tenuous and viscous nature, bridges the gap without deflection. The same is true in *C*, as might be expected from the results in *B*. When a piece of tissue about 1 mm. broad was removed (*D*), a deflection of the mucous streams occurred around the ends of the cut. The mucus which reached the center of the cut was pulled around the ends less rapidly than it arrived, so that it tended to pile up along the cut edge. The adjustment of the mucous flow was not accompanied by any modification of ciliary beat. The direction of effective beat remained exactly as it was before the injury. Nor did the injury cause the adjoining cilia to become quiescent.

These experiments indicate that the ciliated cells do not readily become adapted to a new set of conditions, an adaptation which would seemingly render bacterial contamination around the injured area less probable. The results described are in agreement with those of Hill,[4] who found that cuts across the trachea arranged in alternate fashion stopped the normal passage of mucus.

It may be that some modification in the direction of ciliary beat occurs gradually subsequent to the time when the tissue was studied; however, the experiments of previous investigations furnish evidence against such an opinion. Von Brücke[5] and Merton,[6] who used the roof of the frog's mouth, and Isayama,[7] who studied the trachea of the dog and cat, are all in agreement that the original orientation of ciliary movement is retained in pieces of tissue excised from the host, rotated 180 degrees and reimplanted in the same animal. In the study of the dog reported by Isayama rings 13 to 16 inclusive of the trachea were reversed, and after eleven weeks the polarity of the ciliated epithelium had not changed from that originally held. Von Brücke observed that when the transplant degenerates and is replaced by regenerated tissue from the surrounding epithelium the polarity of the ciliated cells in the new tissue is the same as in its source of origin and does not correspond to that present in the transplant which it replaces. The polarity of the ciliated cells is established early in development in the

4. Hill, L.: The Ciliary Movement of the Trachea Studied in Vitro, Lancet **2**:802 (Oct. 20) 1928.

5. v. Brücke, E. T.: Versuche an ausgeschnittenen und nach einer Drehung um 180° reimplantierten Flimmerschleimhautstücken, Arch. f. d. ges. Physiol. **166**: 45 (Nov. 21) 1916.

6. Merton, H.: Studien über Flimmerbewegung, Arch. f. d. ges. Physiol. **198**:1, 1923.

7. Isayama, S.: Ueber die Flimmerrichtung an verkehrt zur Verheilung gebrachten Abschnitten der Trachea, Ztschr. f. Biol. **82**:155 (Oct. 15) 1924.

embryo of Amblystoma, as shown by Twitty,[8] who stated that the determination of the polarity of ciliated cells slightly precedes the first appearance of cilia.

The mucus (fig. 3*D*), which moves diagonally in relation to the direction of ciliary beat, exerts a mechanical pull against the cilia. Twitty [8] noted that certain mechanical factors were effective in inducing a modification and sometimes a complete reversal in the direction of ciliary beat in the cilia used for locomotion of the embryo of Amblystoma. Reported examples of reversal of ciliary beat have been reviewed by Parker.[9] It is to be noted, however, that the examples in which indisputable ciliary reversal occurs are those in which the cilia, through their phylogenetic history, have acquired this property in order to serve some advantageous function of the organism—for example, to propel the animal away from an undesirable environment or to obtain food.

Another example, in addition to the one shown in figure 3*D*, in which the mechanical element alone failed to cause a reversal of ciliary beat, was given by Lucas and White [10] in their studies of factors producing abnormal distention of the glomeruli in the kidney of Necturus. The normal direction of ciliary movement in the neck is from the glomerulus to the proximal tubule, but in some cases colored fluid was carried through the nephrostome upward into the glomerulus until the latter became distended to several times its normal volume. It was found that temporary blockage of the tubule was the agent responsible, but the important element is the fact that the fluid which was forced upward failed to bring about any reversal of the ciliary beat in the neck. The retrograde flow rendered the movements of the cilia disorganized, but actions that they were able to make were continuously directed downward.

The only example in the animal kingdom coming to my attention in which there is a modification of ciliary polarity in response to injury was observed by Atkins [11] on the gills of certain lamellibranch mollusks. The free pendent edge of the gill contains a groove along which mucous strings bearing entangled microscopic particles of food are transported to the palps and thence to the mouth. Atkins showed that a small crab

8. Twitty, V. C.: Experimental Studies on the Ciliary Action of Amphibian Embryos, J. Exper. Zool. **50**:322 (April) 1928.

9. Parker, G. H.: The Reversal of Ciliary Movement in Metazoans, Am. J. Physiol. **13**:1 (Feb. 1) 1905.

10. Lucas, A. M., and White, H. L.: Factors Responsible for the Abnormal Distention of Glomerular Capsules in the Necturus Kidney, Anat. Rec. **53**:371 (Aug. 25) 1932.

11. Atkins, D.: On Abnormal Conditions of the Gills in Mytilus Edulis: I. On Permanent Natural Reversal of the Frontal Cilia on the Gill Filaments of Mytilus Edulis, J. Marine Biol. A. United Kingdom **16**:919 (May) 1930.

sometimes present in the mantle chamber produces injury to the side of the gill, thus interrupting the line of mucous propulsion downward toward the food groove. This frequently results in the formation of a second groove at the site of injury. After the new groove becomes functional there occurs a reversal in direction of beat of the frontal cilia for varying distances below the new groove. The entire series of changes is gradual and probably requires months. It is her opinion that reversal may occur to some extent in the same ciliated cells which previously beat in the normal direction. Hence, in such instances the reversal is not preceded by degeneration or dedifferentiation of these cells. This point, because of its great clinical importance and for an understanding of the reactions following injury, should be reinvestigated in numerous ciliated epithelia and especially in the respiratory mucous membrane.

CONCLUSIONS

Considering the evidence thus far available in the experiments on invertebrates, amphibians and mammals the following opinions are presented:

1. The ciliated cells do not immediately adjust themselves to new conditions imposed by injuries to the adjacent epithelium.

2. When ciliated tissue is reversed and reimplanted in the same location, surrounded by the same environmental factors of space and equivalent internal forces, the original polarity of implant and surrounding tissue is maintained.

3. Regenerated ciliated epithelium will have the same polarity as it originally held in relation to the tissue from which it originated if the internal and external factors are unchanged.

4. If the environmental or internal factors are altered there may follow a reorientation of ciliary polarity. The new groove along the sides of the gills is an example of a new factor which induces an adaptive change in the adjacent cilia.

Bryant [12] reported that the direction of ciliary movement on the turbinate, in a case in which the condition of the nose necessitated removal of this structure, was toward the anterior nares. This is contradictory to Hilding's [13] observation on the normal nose, that movement

12. Bryant, W. S.: An Experiment to Prove that the Cilia of the Human Nose Waft Toward the Anterior Nares, Am. J. Physiol. **33**:430 (March 2) 1914.

13. Hilding, A.: Ciliary Activity and Course of Secretion of the Nose, Proc. Staff Meet., Mayo Clin. **6**:285 (May 13) 1931; The Physiology of Drainage of Nasal Mucus: I. The Flow of the Mucus Currents Through the Drainage System of the Nasal Mucosa and Its Relation to Ciliary Activity, Arch. Otolaryng. **15**:92 (Jan.) 1932.

is toward the choanae. If point 4 is found to hold generally, the difference may perhaps be explained. The factor effective in this case would be the decreased size of the space in which the cilia must move the mucous stream owing to the abnormal structure of the nose and the membranes. It was pointed out by Lucas [14] that when the space between opposed ciliated surfaces is small the cilia beat toward the nearest larger cavity without regard to the axis of the nose. Additional evidence of the validity of this principle will be presented in a forthcoming publication.

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14. Lucas, A. M.: Direction of Ciliary Movement in the Nasal Cavity of Macacus Rhesus (Desm.): A Preliminary Report, Tr. Am. Laryng., Rhin. & Otol. Soc. 1931, p. 172; The Nasal Cavity and Direction of Fluid by Ciliary Movement in Macacus Rhesus (Desm.), Am. J. Anat. **50**:141 (March 15) 1932.

Clinical Notes

AN EYE RETRACTOR AND A SUTURE-CARRIER KNOT-TIER FOR THE EXTERNAL PANSINUS OPERATION

ROMEO A. LUONGO, M.D., PHILADELPHIA

THE EYE RETRACTOR

This instrument is a modification of the Ferris Smith retractor, used for the external operation on the ethmosphenofrontal group of sinuses with ligation of the anterior ethmoidal artery, as described by Sewall [1] and Ferris Smith.[2]

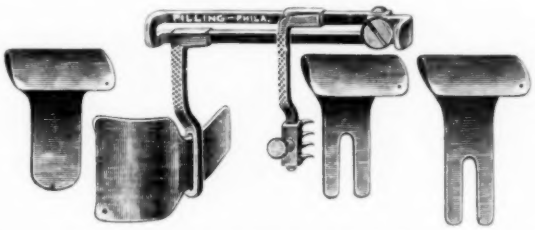

Fig. 1.—The retractor and the four blades. The arm to the left has a slot for the blades, and the arm to the right ends in a square piece on which a sleeve-like appliance with teeth is slipped and fixed with a set screw. To reverse: Take the blade out, slide the left arm to the right and the right arm to the left and turn the teeth around.

Fig. 2.—The suture-carrier knot-tier.

The retractor can be made to fit both on the right and on the left side by switching the arms around and reversing the teeth (fig. 1).

Each blade has an eye shield which will protect the eye, making the stitching of the lids unnecessary. There are four blades, two slotted and two plain. The slotted ones are used for ligation of the artery and the plain ones for better protection of the orbital contents. If the orbital periosteum is accidentally injured, exposing the orbital fat, the solid blade will keep this fat out of the field, and it

Read (by invitation) at a meeting of the American Laryngological Association, Washington, D. C., May 8, 1933.

1. Sewall, E. C.: External Operation on the Ethmosphenoid-Frontal Group of Sinuses Under Local Anesthesia, Arch. Otolaryng. **4**:377 (Nov.) 1926.

2. Smith, Ferris: Chronic Sinus Disease, J. A. M. A. **100**:402 (Feb. 11) 1933.

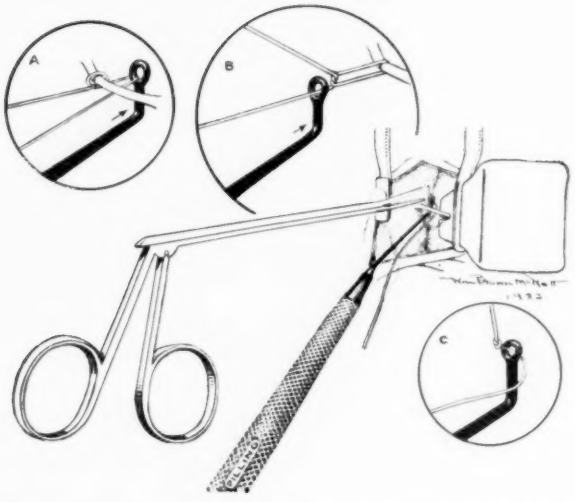

Fig. 3.—The tying of the anterior ethmoidal artery with the suture-carrier knot-tier. *C* represents the threaded instrument, showing the knot and the free end of the catgut above it (the knot will become engaged into the ring by pulling on the other end of the catgut). The central figure shows the instrument in place around the artery with the free end of the catgut (beyond the knot which is already engaged into the ring) ready to be grasped by a Hartmann alligator forceps. *B* represents the process of pushing the knot toward the artery. *A* represents the process of tightening the knot around the artery.

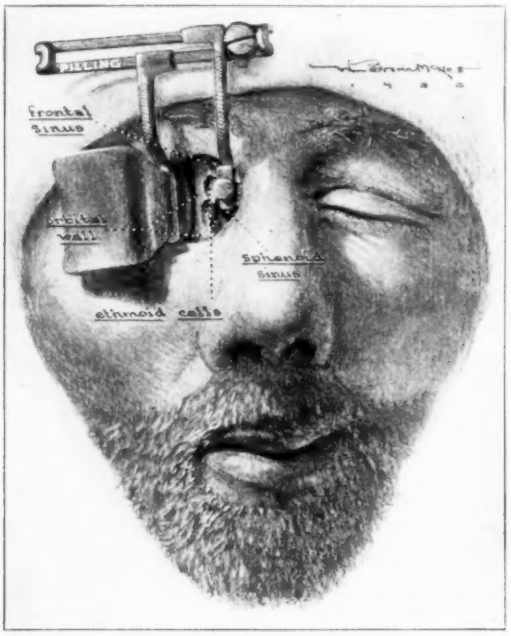

Fig. 4.—The retractor in place.

will prevent the infected material from the nose and sinuses from coming in immediate contact with the contents of the eye through the periosteal opening. After the ligation of the artery, I change from the slotted to the solid blade in order to protect the ligated stump of the artery, which often will persistently protrude through the slot, interfering with the view and being exposed to a possible injury by the instruments during the operation.

Prying with the point of a hemostat inserted between the arms will facilitate the opening of the instrument. A hemostat is also helpful in the closing of the instrument.

In opening and closing the instrument, force should be applied as near as possible to the transverse bars on which the arms slide.

THE SUTURE-CARRIER KNOT-TIER

Ligation of the anterior ethmoidal artery can be done with this instrument alone. The catgut is threaded through the small ring at the end of the instrument and then a knot is made about a quarter of an inch (0.6 cm.) from the end of the catgut (fig. 3 *C*). When the catgut is stretched, the knot will become engaged into the ring and the stiffness of the catgut will keep the free end (beyond the knot) in such a position that it can be easily grasped with a small hemostat or forceps. The knotted end of the catgut is pulled out while the instrument is kept in place (the ring will protect the artery from the sawlike motion of the catgut against the vessel during this pulling). Without being unthreaded, the instrument is then withdrawn, and a knot is made between the ring and the artery. This knot is pushed toward the artery and tightened as illustrated in *A* and *B* of figure 3. Subsequent knots are made and tied in the same manner.

This instrument can also be used as a probe for intranasal as well as for extranasal work.

These instruments are made by the G. P. Pilling Co.

1713 Pine Street.

PLASTIC OPERATION FOR PROTRUDING EARS

HENRY M. GOODYEAR, M.D., CINCINNATI

This paper is concerned with protruding ears that are especially objectionable because of the bending out or lopping of the upper portion and the protrusion of the lobe, accompanied by a general displacement forward of the entire ear.

Most ears with these characteristics are unusually large, and when they are brought back closer to the head by removal of the soft tissues as I shall describe, the lopping of the upper portion remains, and the lobe is still in an abnormally prominent position.

In the present case, on careful analysis I noted that the absence of an anthelix was one of the most disfiguring factors, giving a "dog ear" appearance and accentuating the size by presenting an unbroken surface.

Read before the Cincinnati Oto-Laryngological Society, April 25, 1933.

OPERATION

First, the general forward position of the ear was corrected in the manner described by Kerrison,[1] except that the incisions were carried to the extreme upper and lower attachments of the ear, and the deep structures, as much as possible, were included in the sutures.

With a tooth thumb forceps the skin was grasped on the back of the ear and the auricle brought against the scalp. When the ear was in approximately normal position, the contact points were nicked (fig. 1, *X*, *X*). An elliptic incision was then made down to the perichondrium on the auricle and to the periosteum over the mastoid (fig. 1 *A*). Silk sutures were used to close this denuded area, care being taken to include the deep tissues.

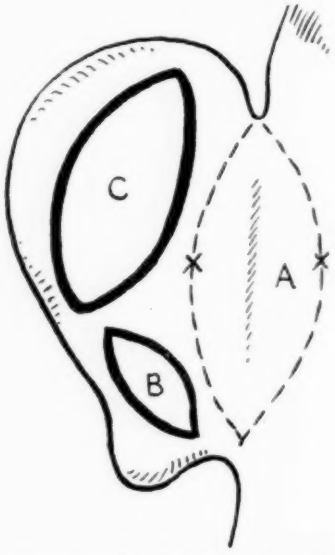

Fig. 1.—*A*, area of tissue removed to bring the general position of the ear nearer the head. *B*, outline of tissue removed to correct the outward displacement of the lobe and lower portion of the auricle. *C*, outline of tissue removed to form an anthelix.

This operation brought the general position of the auricle near the head, but the disfiguring curving out of the upper and lower portions remained.

The lower outer curvature was corrected by a wide elliptic incision as indicated in figure 1 *B*.

The third step, and the one of the greatest interest, was that of correcting the lop and reducing the size of the upper portion of the auricle. I decided that if I could make an anthelix, not only would the ear be reduced in size, but a normal-appearing ear would be approximated. To accomplish this a long elliptic resection was made (fig. 1 *C*) over the area corresponding to a normal anthelix. The resection was carried to the perichondrium, but no cartilage was removed. On

1. Kerrison, P. D.: Diseases of the Ear, Philadelphia, J. B. Lippincott Company, 1923.

closing this third incision, the appearance of the whole ear was immediately transformed; in fact, the improvement was astounding.

I should like to caution here that rather a bold and wide excision of tissue is necessary to produce a good anthelix.

Tincture of merthiolate was used throughout, and all sutures were of silk. Several layers of gauze were placed behind the ear, and the outer surface was packed with cotton, conforming to the contour.

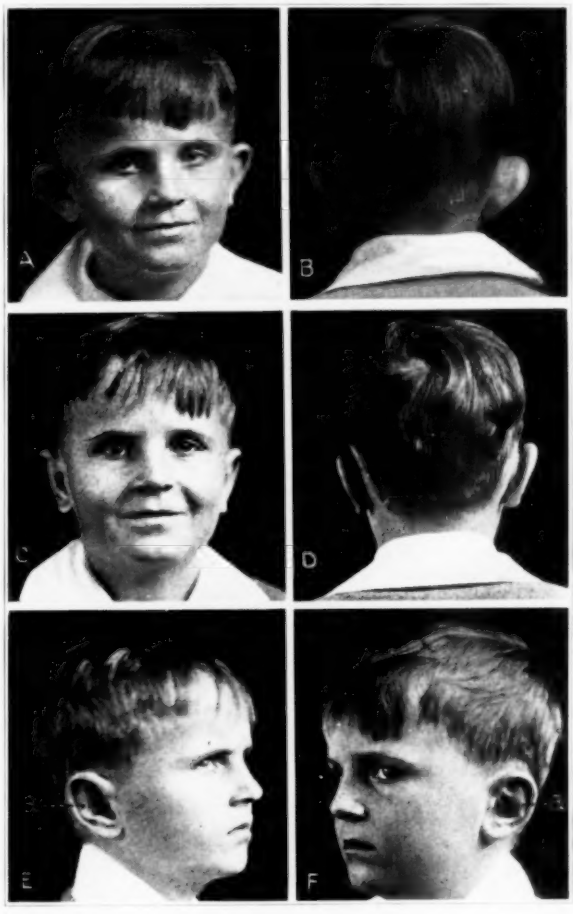

Fig. 2.—Appearance of patient (aged 8 years): *A*, *B*, before operation; *C*, *D*, corresponding views after operation; *E*, *F*, after operation; note the well formed anthelix (*a*).

I did not disturb the bandages for one week except to peer under them for evidence of infection. At the end of the week the dressing was removed. Tincture of merthiolate was again applied to the back of the ears, and the bandage was replaced.

On the tenth day all sutures were removed, but the bandage was worn for three weeks.

SUMMARY

The absence of the anthelix is the most deforming characteristic of a lop-ear.

The apparent size of the ear is greatly reduced by the construction of an anthelix.

The entire operation is done without incision of the auricular cartilage, thus minimizing the dangers of a chondritis.

556 Doctors' Building.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

PLASTIC SURGERY

J. EASTMAN SHEEHAN, M.D.
Professor of Surgery, Columbia University
NEW YORK

What appeals to one in "The Operative Story of Cleft Palate" [1] is the air of conscious and justified authority with which its author, Dorrance, invests it. He has reviewed all that has been written on the subject and has subjected the devices that bear great names to the test of his own knowledge and experience. The result is a monumental compendium.

Equally illuminating, though in another manner, is the contribution of Gillies to the clinical detail of dealing with the secondary deformities associated with harelip.[2] That these conditions are due to malformations which continue or develop after the original closure is well known, and whereas in the main they may be assigned to asymmetry of the nasal alae and to imperfection in the form of the upper lip, there is a vast variety in the irregularities presented, with corresponding drafts on the surgical ingenuity by which correction can be effected. Gillies demonstrated excellently the possibilities of attainment when the resources of reparative surgery and dental prosthetic skill are combined under the most competent direction.

It has been urged in these reviews that approachable cancer is one of the major interests of plastic surgery. This view finds confirmation in two reports of the present year. In one Trant [3] provided conclusive evidence of the superior results to be obtained from excision by electric coagulation when one is dealing with small growths on the face, and in the other Smith [4] demonstrated what can be done in restoring the form of the lip after an excision for malignancy. Judicious employment of skin grafts in the double thickness necessary for the lining and cover

1. Dorrance, G. M.: The Operative Story of Cleft Palate, Philadelphia, W. B. Saunders Company, 1933.

2. Gillies, Harold, and Kilner, T. P.: Hare Lip: Operations for Correction of Secondary Deformities, Lancet **2**:1369, 1933.

3. Trant, E. F., and Tolemach, J. A.: Squamous Cell Epithelioma of the Skin of the Face, New York State J. Med. **33**:875 (July 15) 1933.

4. Smith, Ferris: Surgical Management of Lip Malignancies, Surg., Gynec. & Obst. **56**:782, 1933.

has for a result the practically complete elimination of the defect and therefore of the esthetic blemish.

As to research in this field, there is the account by Davis [5] of his experiments at Johns Hopkins University in the treatment of old, unhealed burns. Such cases, as he carefully points out, are outside the range of casual practice. They call for a hospitalization of many months. Cases of this sort have to do with surfaces unfit for covering with skin by ordinary methods because of their extent and because of infection which cannot be cleared up without risks too great to be imposed on the enfeebled patients. In old, unhealed burns is an admirable field for the multiple emplacement of the small, thick grafts from which epithelization may and does proceed in the face of these disabilities.

Attention has heretofore been called, not to the unwelcome and unanticipated developments arising from the progress in radiotherapy—the utilization of forces whose efficacy has been demonstrated before their mysteries have been unveiled—but to reparative surgery as practically the only resource in such calamities. In this field surgeons are again indebted to Gillies [6] both for the indications afforded of the variety of the inflammations and of the resultant distortion and dysfunction, and for the evidence that the major tragedies which have become all too familiar are not necessarily inevitable. Valuable also in this connection is Kaplan's [7] discussion of the use of radon seeds and its attendant dangers. These emanations from radium have been shown to possess the properties of this element, including those of its dangerous rays. A good effect is observed, for example, when the emanations are allowed to stimulate the opposing edges of refractory skin for a short period, but when the radon seeds are left in situ for the longer period essential to malignant conditions, the dangerous rays exert their influence. It is necessary to find a medium that will allow passage of the curative and not of the destructive rays.

In the field of anesthesia, emphasis this year appears to be on the extension of the avertin method. Shipway found it adaptable to operations in the upper respiratory tract; [8] others have been experimenting with it in conditions of the nose and throat, and there appears to be a general disposition to use it when the desire is to eliminate specific apprehension (as in goiter) or the nervousness of young children.

5. Davis, J. S., and Killowski, E. A.: The Treatment of Old Unhealed Burns, Ann. Surg. **97**:683 (May) 1933.

6. Gillies, H. D., and McIndoe, A. H.: Plastic Surgery in Chronic Radiodermatitis and Radionecrosis, Brit. J. Radiol. **6**:132 (March) 1933.

7. Kaplan, I. J.: The Use and Value of Gold Radon Seeds, J. A. M. A. **100**:1765 (June 3) 1933.

8. Shipway, Francis: A Note on Avertin Anaesthesia in Operations on the Upper Respiratory Tract, J. Laryng. & Otol. **47**:389 (June) 1933.

Those who agree as to its advantages nevertheless insist that there are dangers to be guarded against. This phase of the subject is well stated in Pearse's [9] complete study.

So much in the field of reparative surgery has to do with success in the transfer of skin grafts that those who are immediately concerned cannot afford to neglect any intelligent discussion on the properties of the covering of the body. Whether it is to be rated as a separate organ [10] and placed in a category with organs more familiarly described as separate is a question that may be left to those who have been advocating this view, but any one who speculates as to why one skin graft does well and another, made under apparently like conditions, does not, endeavors to find a test by which the adequacy of the tissue can be determined in advance, and a means by which any deficiency can be made good. Experiments in my own service indicate that the skin's oxygen content is an important factor, and that routine procedures may unduly lower the oxygen content if applied at the wrong time. Curiously enough, the biochemical experiments made in this connection strongly suggest the influence of viosterol and cholesterol, recently declared of great importance, and of the sulphur content. The healing of wounds must also be a matter of unceasing interest, since esthetic results in exposed areas depend so much on it. One must be grateful for the study by Du Mortier [11] who found a progressively increasing resistance to infection, from a maximum liability at six hours to a minimum at five days. How to apply this knowledge to the production of a minimum scar will always be a problem for the reparative surgeon.

In the main, results favor the use of reparative tissues taken from within the body or from its surface rather than those in any sense foreign to it. When there is a question of using paraffin, ivory or, in certain cases, the precious metals, the time for doubt has long been passed, for the physician is in the presence of disaster. But there is nothing so unscientific as to condemn without examination, and those who assert that there is merit in the use of bone for the repair of defects of the skull [12] and those who hold that ox fascia will serve the purpose and prevent the discomfort attending the acquisition of fascia from the patient [13] are entitled to a respectful hearing before their claims are dismissed.

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9. Pearse, H. J., Jr., and Wiedrick, M. M.: Avertin Anesthesia, New York State J. Med. **33**:385, 1933.

10. Garrison, F. H.: The Skin as a Functional Organ of the Body, Bull. New York Acad. Med. **9**:417 (July) 1933.

11. Du Mortier, J. J.: The Resistance of Healing Wounds to Infection, Surg., Gynec. & Obst. **56**:762 (April) 1933.

12. Pankratiev, B. E.: Dead Bone Grafts to Repair Skull Defects, Ann. Surg. **97**:321 (March) 1933.

13. Glasser, S. T.: Ox Fascia Grafts, Am. J. Surg. **19**:542 (March) 1933.

Abstracts from Current Literature

Ear

MÉNIÈRE'S DISEASE: DIAGNOSIS AND TREATMENT. C. C. COLEMAN and J. G. LYERLY, Arch. Neurol. & Psychiat. **29**:522 (March) 1933.

The authors report excellent results in the treatment of eight patients with Ménière's disease by intracranial section of the eighth nerve.

BEVERLY, Chicago. [AM. J. DIS. CHILD.]

CONGENITAL MALFORMATION OF THE AURICLE ASSOCIATED WITH PARALYSIS OF THE FACIAL NERVE. CLAIN FANNING GELSTON and EMMETT E. SAPPINGTON, Arch. Pediat. **49**:730 (Nov.) 1932.

Gelston and Sappington review the literature on congenital malformation of the auricle associated with paralysis of the facial nerve and report a case. A review of the embryologic development of the external ear is presented in detail, showing how variations in embryologic development result in such deformities.

WADDELL, JR., University, Va. [AM. J. DIS. CHILD.]

MENINGEAL LESIONS COMPLICATING AURAL INFECTIONS. M. R. MOBLEY and J. P. PRICE, J. South Carolina M. A. **29**:4 (Jan.) 1933.

Mobley and Price divide meningitis into four groups: (1) serous, (2) lymphocytic and (3) polymorphonuclear or purulent meningitis and (4) meningism. Only three of the four types are found in meningitis of otitic origin, and, in order of frequency, the purulent type, the serous type and, in rare cases, meningism are found. In some cases in which patients show purulent meningitis, repeated cultures are sterile.

The symptoms are nausea, headache and slight stiffness of the neck, accompanied by a slight rise in temperature and an increase in the number of leukocytes in the blood. The signs may vary from slight to severe. As long as the condition is localized it is curable, and the only treatment is surgical, which consists of mastoidectomy. To offer proper drainage, repeated lumbar punctures are advised. Two cases in which treatment was successful were observed by the authors.

ORSTON, JR., Columbia, S. C. [AM. J. DIS. CHILD.]

ACUTE OTITIS MEDIA IN INFANCY. N. ASHERSON, Arch. Dis. Childhood **7**:159 (June) 1932.

The author (a surgeon for diseases of the ear, nose and throat), in a study of acute otitis media in infants, describes the anatomy of the middle ear and the mastoid. He reports several cases of purulent otitis media in new-born infants. In these cases the symptoms are usually: (*a*) fever of unknown origin, (*b*) obvious earache, (*c*) a discharge from the ear or (*d*) signs suggestive of meningitis. The tympanic membrane may appear to be normal, and still there may be pus deep in the middle ear. In cases of acute otitis media in which the temperature is 100 F., myringotomy should be performed. In cases of infantile gastro-enteritis, the ears should be observed frequently. If myringotomy reveals pus, the antrum should be drained.

KELLY, Milwaukee. [AM. J. DIS. CHILD.]

NASAL DISCHARGE OF CEREBROSPINAL FLUID, LASTING FOR YEARS. F. J. COLLET, Rev. d'oto-neuro-opht. **10**:660 (Nov.) 1932.

This contribution is a detailed report of the case of a man who, following a fall on the head, had a flow of cerebrospinal fluid from the nose that lasted nearly

three years. The flow was small in quantity and more or less intermittent, and was accompanied by headaches, epileptic attacks and anosmia. It was assumed that the patient had suffered a fracture of the cribriform plate, although it is known that fracture of the petrous bone without tympanal rupture can cause a leakage of the fluid through the eustachian tube and thence through the nose. Anosmia may be caused by tearing of the meninges and compression of the olfactory nerves by the consequent hemorrhage or by tearing of the olfactory filaments themselves. Anosmia is a frequent complication of cranial traumatism, especially when the force is applied to the occiput or forehead. It is noteworthy that no serious consequences have resulted in this case from the prolonged flow. Of sixty patients with cranial fractures accompanied by nasal loss of cerebrospinal fluid reported by Schoenbauer and Brunner, eighteen died. This does not take into account those who may die from meningitis after the cure is apparently complete. DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

THE DIAGNOSTIC SIGNIFICANCE OF THE HYDROGEN ION CONCENTRATION OF THE CEREBROSPINAL FLUID OF PATIENTS WITH OTOLOGIC DISEASE. K. LÖWY, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:180 (March 21) 1933.

Löwy states that the normal p_H value (about 7.45) of the spinal fluid is generally unaltered in intracranial complications due to otitis. Only severe purulent meningitis is usually accompanied by a decrease of the p_H to about 7.2. No early diagnostic value can be attributed to these p_H measurements. The constancy of these values is based on the buffering of the cerebrospinal fluid. Whether there is a relation between the occurrence of certain neurologic symptoms and the increased acidity of the spinal fluid cannot be decided on the basis of present investigations.

EDITOR'S ABSTRACT.

A POSITIVE BABINSKI SIGN IN MÉNIÈRE'S SYNDROME. S. BERGGREN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:273 (April 4) 1933.

Berggren reports that he has observed the appearance of a positive Babinski sign in four patients with Ménière's syndrome. In all cases the patellar reflexes were lively without being exaggerated. Aside from the vestibular symptoms existing in all cases and the cochlear symptoms present in three, neurologic symptoms were demonstrable in only one case. In this case the positive Babinski sign, while transient, was of rather long duration and appeared of serious prognostic significance. In the other cases the Babinski sign could be only demonstrated during an attack or during and immediately after an attack. The only other pyramidal symptoms were a slight transitory paralysis in the case in which the Babinski sign was most pronounced. While the author does not know of any other analogous cases, a positive Babinski sign is known to occur in certain cases in which there is no organic lesion of the pyramidal tract, but merely a transitory disturbance of function which must be due to exogenous or endogenous poisons (strychnine poisoning; comatose sleep produced by morphine, barbital and similar drugs; comatose states caused by metabolic disturbances). Ménière's syndrome involves temporary disturbances of compensation and the influence of various factors, such as fatigue and exogenous or endogenous intoxication, with complete compensation later. It appears that the same factors which are at the base of Ménière's syndrome in exceptional cases suffice to produce a transitory and reparable functional disturbance of the pyramidal tract.

EDITOR'S ABSTRACT.

PATHOGENESIS, DIAGNOSIS AND TREATMENT OF DISEASES WITH ATTACKS OF VESTIBULAR VERTIGO (ACOUSTIC CRISES). J. KOCH, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:314 (April 4) 1933.

Koch studied thirty-eight patients with widely varying symptoms all of whom had in common the predominating symptom of attacks of vestibular vertigo.

Patients with an organic space-limiting disease of the brain or with genuine epilepsy were not included. In three cases the attacks were of the real Ménière type; in twenty-two there were cochlear and vestibular disturbances, and in thirteen there were only vestibular symptoms. As all the symptoms are produced by disturbances somewhere in the path of the acoustic nerve, the author thinks that the designation of "acoustic crisis," suggested by Grahe and Kobrak for all cases of vestibular vertigo, is appropriate and should replace the terms Ménière's, pseudo-Ménière's or Bárány's syndrome. His examinations indicated that all the acoustic crises observed by him were the results of organic or neurotic vascular disease which led to disturbances of the blood supply in the labyrinth or the central portions of the acoustic nerve. The results of cochlear examination, the contrasting reactions in vestibular examination, the unilateral absence of the galvanic reaction and certain changes in the sense of position indicated that in thirty-two cases the seat of the disturbance was in the central portion of the acoustic nerve. The causes of vasomotor disturbance in the path of the acoustic nerve included alveolar pyorrhea, encephalitis, epilepsy, food allergy, nicotine intoxication, polycythemia, disturbances of sexual and spiritual life and mechanical trauma of the skull. In two cases a relation could be discovered between acoustic crisis and chronic tonsillitis, and in another case between acoustic crisis and glaucoma. Pharmacologic tolerance tests and Muck's epinephrine-probe test were employed in the study of the sympathetic nervous system. Pronounced vagotonia or sympathicotonia was found in few cases; in most cases there were mixed disturbances. Causal therapy was most effective in curing the acoustic crises. In cases of undetermined etiology an improvement or cure of the vestibular disturbances and tinnitus was obtained by symptomatic therapy. The baryecoia or deafness was not relieved by causal or by symptomatic therapy.

EDITOR'S ABSTRACT.

INJURIES TO THE MECHANISM OF HEARING AND EQUILIBRIUM PRODUCED BY A STRONG ELECTRIC CURRENT. R. PERWITZSCHKY, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:345 (April 4) 1933.

Perwitzschky presents a tabular review of the twenty-four cases reported in the literature in which injury to the mechanism of hearing and equilibrium was produced by a strong electric current and adds an observation of his own. Injuries may be early or late (up to two years). The form and the degree of the injury are in no way dependent on the type of current or the tension. The point at which the current enters and leaves is also insignificant. In most of the cases there was a labyrinthine baryecoia. The hearing was impaired more often than the equilibrium. Only in one case was an isolated disturbance of the vestibular apparatus demonstrable. A decrease in the disturbance of hearing may occur, but an increase is much more frequent. The nature and location of the disturbance can only be guessed at. It is thought that the injuries are located in the nuclei of the medulla.

EDITOR'S ABSTRACT.

THE NORMAL ANATOMY OF THE OTIC CAPSULE. M. MEYER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **34**:3 (May 6) 1933.

Meyer states that the normal anatomy of the otic capsule is of interest because of the basic difference of the osseous structure of this part of the skeleton and of its mode of development from those of any other bone in the body, and because only an exact knowledge of the normal structure permits recognition of pathologic deviations from it. After describing in detail the normal anatomic structure of the otic capsule in man and its development, he discusses separately cartilaginous rests, fissures and the ossicles. He also studied the normal anatomy of the otic capsule in laboratory animals, and concluded that monkeys and dogs are well suited for experimental purposes because the structure of the otic capsule in these animals is analogous to that of human beings. In cats, rabbits and guinea-pigs the structure of the capsule is basically similar to that of man, but there are differences in details

which demand great caution in the comparison of experimental results. Rats, mice and chickens must be excluded in comparative studies because the osseous structure of the otic capsule in these animals is totally different from that in human beings. In a concluding section on the functional structure of the petrosa and the otic capsule the author states that from a consideration of all the facts concerning the entire structure of the petrosa, the deviation from the normal in the structures of the otic capsule on the one hand and the rest of the petrosa, on the other, and the striking similarity of these conditions in human beings and certain mammals, one can assume that the mechanical forces which act on the whole petrous bone are also exerted on its compact portion, the otic capsule. Hereditary factors of unknown cause are responsible for the difference in the reaction of the otic capsule and of the rest of the petrosa to the existing mechanical stimuli.

EDITOR'S ABSTRACT.

PATHOLOGIC ANATOMY OF THE OTIC CAPSULE AND ITS CLINICAL SIGNIFICANCE. F. R. NAGER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **34**:72 (May 6) 1933.

Nager discusses most of the bone diseases in which changes in the otic capsule have been observed by him or by other investigators. He includes osteoporosis, osteolysis, osteosclerosis, inflammatory changes of the otic capsule, congenital syphilis, xanthomatosis, ochronosis of the temporal bone, chloroma of the temporal bone, healing processes in injury of the capsule, chondrodystrophy, osteogenesis imperfecta, rickets, osteomalacia, cretinism, athyreosis, mongolian idiocy, oxycephaly, osteodystrophia fibrosa, osteitis deformans, otosclerosis and experimental changes of the otic capsule. Osseous diseases of the otic capsule assume a special character. While severe destructive processes may totally destroy the osseous tissue, most skeletal diseases occurring after birth start in the outer layer and remain localized there. Only in diseases leading to severe regeneration of bone, such as fibrous osteodystrophy, are the inner layers of the capsule involved. The unique biologic position of the capsule is also demonstrated in its reaction to injuries, especially fractures; the endochondral layer of the capsule, in contrast to all other bones, does not possess the capacity for bone regeneration in injury. Diseases of the bones occurring in fetal life do not spare the endochondral layer; they cause malformation of tissues and result in functional disturbances of the cartilage-producing and bone-producing centers. The otic capsule is particularly suitable for osteopathologic study because the reformed bone always exhibits a more mature tissue than the stroma, which permanently retains cartilaginous rests and unlamellated bone. The processes of ossification can be well studied in the otic capsule. The main problem of the osteopathology of the otic capsule, otosclerosis, is not yet solved.

EDITOR'S ABSTRACT.

Pharynx

DO PATHOGENIC MICRO-ORGANISMS GET INTO THE CIRCULATION DURING TONSILLECTOMY. E. WIRTH, Deutsche med. Wchnschr. **58**:1755 (Nov. 4) 1932.

It is well known that sometimes after tonsillectomy there are recurrences of previously existing systemic diseases, such as polyarthritis, nephritis or endocarditis. The explanation of this observation by some writers is that pathogenic organisms and toxins might enter the circulation in connection with the removal of the tonsils. This contamination of the blood stream occurs more often if the tonsils are acutely inflamed before removal.

The author made five cultures of blood for each of forty-five patients before and after removing their tonsils. Fifteen did not have acute inflammation of the tonsils at the time of operation. Five patients had recovered from an acute attack, from one to three weeks prior to the operation. In twenty cases, besides recurrent sore throat, general systemic disease was also present.

In all of the forty-five patients the five cultures of the blood were negative.

KERESZTURI, New York. [AM. J. DIS. CHILD.]

HEMORRHAGES FOLLOWING TONSILLAR DISEASE. W. BRAUER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:558 (March 20) 1933.

Brauer gives a comprehensive discussion of hemorrhages following tonsillar disease, based on the literature and on nine personal observations. Such hemorrhages occur chiefly in inflammations in the peritonsillar tissue. It is often difficult to determine which vessel has been eroded. In thirty-three of the seventy-two cases in the literature, the hemorrhages were healed; in seven cases spontaneously or by tamponade, in three by tonsillectomy, in ten by ligature of the external carotid artery, and in thirteen by ligature of the common carotid. Of the thirty-nine patients who died, thirty received no treatment. While these figures show the value of ligation in severe cases, the author does not think that it is indicated in every case of hemorrhage but insists on hospital supervision, so that no time will be lost if surgical intervention becomes necessary. Early incision of abscesses is a prophylactic measure. General rules for therapy are discussed. In slight hemorrhages tamponade may be employed. If the exact location is known, acupression, sometimes after tonsillectomy, may be used; in other cases tonsillectomy alone stops bleeding. Such hemorrhages usually come from branches of the external carotid artery. Severe hemorrhages demand ligation of the vessel concerned. If compression of the external carotid stops the hemorrhage, only this artery need be ligated. If compression does not stop the bleeding or decrease it materially, the internal carotid is involved, and the common carotid must be ligated as branches of the external carotid usually terminate in the abscess also and ligation of the internal carotid alone would not suffice. If ligation is performed after the hemorrhage has stopped, the external carotid is ligated and a loose ligature applied to the common carotid (its end issuing from the wound) which may be constricted if necessary. After ligation of the vessels, the phlegmon in the pharynx must be incised. In rare cases in which ligature of the common carotid is not effective because of hemorrhage from the peripheral vascular region or anastomoses of the other side, tight tamponade must be used. If there is the slightest cause for suspecting an aneurysm, a loose ligature must be placed around the common carotid before the phlegmon is incised so that a fatal hemorrhage may be prevented. For small venous hemorrhages the same therapy as for small arterial hemorrhages is indicated. Tight tamponade will often stop hemorrhage even from the internal jugular vein. Hemorrhage from ulcerations in the tonsils and hemorrhage from the tonsils due to general disease are briefly discussed.

EDITOR'S ABSTRACT.

ASCENDING INFECTION OF THE ENDOCRANIUM WITH TONSILLOGENOUS PHARYNGEAL PHLEGMON. K. VOGEL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:4 (April 12) 1933.

This unusual spread of infection was observed by Vogel in three cases which he reports. In two of the cases, following tonsillitis, a peritonsillar abscess developed, accompanied by septicemic symptoms, a pronounced painful swelling of the temporal region and meningitis which terminated fatally. In the third case the disease started with osteomyelitis of the right superior maxilla, the hard palate and finally the nasal septum. This was followed by swelling of the left side of the soft palate with thickening of the anterior and posterior palatine arch. The left temporal region was swollen, and symptoms of meningitis and paralysis of the abducens nerve appeared. The patient recovered from these symptoms in three weeks, but died later of an abscess of the left frontal lobe. A simple phlegmonous spread of the infection from the peritonsillar region is contraindicated by the absence of purulent infiltration in the parapharyngeal space. In all three cases thrombosis of part of the cervical veins was found. In the first case it occurred in the branches of the right anterior and posterior facial veins and the veins of the cavernous and intercavernous sinuses. In the second case there was thrombophlebitis of the left jugular vein and the veins of the left pterygopalatine fossa up to the cavernous sinus, the

left jugular bulb and the sigmoid sinus. In the third case, thrombophlebitic changes were found in the partially excised facial vein. The author calls attention to the characteristic painful swelling in the temporal region which probably results from infection of the pterygoid plexus and the venae temporales profundae communicating with it. This ominous swelling is an early manifestation of the ascending path of the thrombophlebitic infection, which cannot be stopped by ligation and probably always terminates fatally, as in the cases reported. EDITOR'S ABSTRACT.

TONSILLITIS WITH NEPHRITIS AND PERITONSILLAR ABSCESS DESPITE TONSILLECTOMY. V. HINSBERG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:77 (April 12) 1933.

Hinsberg reports one personal observation of an acute infection of the "tonsillar region" accompanied by nephritis several years after tonsillectomy and four cases of peritonsillar abscesses occurring despite tonsillectomy, two of which are personal observations while two are cases from the literature. These four cases show that despite tonsillectomy, and sometimes several years later, an abscess may be formed in the region of the scar which presents the typical picture of a peritonsillar abscess and which may heal spontaneously or after incision or may lead to severe sepsis and death. In one of the two cases from the literature a small part of the superior tonsillar pole remained after operation; in the other case also this probably occurred. In one of the author's cases a piece of tonsillar tissue half the size of a pea was found in the scar; in the other case such a fragment appeared to have remained at the lower pole. These tonsillar remnants are probably to be considered as the focus for the abscess formation. How infection occurred is not certain. The practical interest of these observations lies in the fact that abscess formation may occur even after tonsillectomy and in the warning contained in them to avoid leaving even the smallest tonsillar remnants when performing tonsillectomy, especially on the upper pole. EDITOR'S ABSTRACT.

Larynx

MULTICENTRAL ORIGIN OF CARCINOMA OF THE VOCAL CORD. C. E. BENJAMINS, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:264 (April 4) 1933.

Benjamins reports a case of carcinoma of the vocal cord in which the histologic changes indicate that a tumor which spreads evenly over the entire cord, as is sometimes observed, may have a multicentral origin. In this case only a slight nodular thickening was visible at one point in the right vocal cord, and deviation of the cord was slight, but in phonation it did not follow the movement of the right arytenoid muscle, indicating infiltration of the muscles of the cord. There was also a thickening of the false vocal cord. After biopsy on the nodular portion of the vocal cord the diagnosis was squamous cell carcinoma. Hemilaryngectomy with preservation of the cricoid cartilage was performed. Histologic examination disclosed small separate carcinomatous centers on the surface of the vocal cord. One of these was found to be definitely connected with a scirrhus located deep in the muscular and connective tissue layer, but most of the small carcinomas were not connected with the scirrhus. The author thinks that if the disease had continued longer, the originally separate and macroscopically invisible carcinomas would have merged and the laryngoscopic picture would have been that of a tumor spreading evenly over the vocal cord. The case demonstrates that carcinoma of the vocal cord can develop from multiple malignant centers. So far as therapy is concerned, the case shows that the occurrence of a single macroscopically visible tumor is not a guarantee against the occurrence of malignant degeneration at other spots. Extensive incision is therefore advisable, especially if the movement of the vocal cord is restricted. EDITOR'S ABSTRACT.

PATHOLOGY AND THERAPY IN UNILATERAL PARALYSIS OF THE RECURRENT NERVE FROM THE STANDPOINT OF PHONIATRICS. R. IMHOFER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:26 (March) 1933.

Contrary to some other authorities, Imhofer thinks that the vocal cord possesses no independent motion after paralysis of the recurrent nerve; its only motion is that which is imparted to it by the impact of the normal vocal cord. However, the loss of the vibratory function does not necessarily produce a disturbance of phonation. If the paralyzed vocal cord approaches sufficiently near the median line and attains a state of tension sufficient to offer the required resistance to the current of air, satisfactory phonation can be achieved without a vibratory function or adjustment to the vibration of the other side. In fact, it is better that the vocal cord should not vibrate or should undergo only enforced contact vibration than that it should cause fluttering or an unclear tone by independent vibrations which differ from those on the other side or change during a tone. To bring this about, the author applies two small cupping glasses to the thyroid cartilage and aspirates them until they are half filled with the skin and soft parts. If the clamped tubes are given a pull during fixation of the cricoid cartilage, the thyroid cartilage turns forward as it does under the influence of the cricothyroid muscle and a tension of the vocal cord results. In this way an asymmetrical influence can be exerted on the vocal cords. Vocal exercises are performed during this procedure. During pulling on the tubes, the voice immediately becomes clearer; it also becomes higher, but this is moderated in time. Phoniatric therapy should be instituted as soon as possible after injury to the recurrent nerve to prevent atrophy and excavation, especially after thyroidectomy. The therapy described cannot be applied until complete closing of the incision and a resistant scar formation are attained.

EDITOR'S ABSTRACT.

Nose

STUDIES IN CHRONIC SINUSITIS IN CHILDREN. J. S. UHR and J. W. PUGH, J. Pediat. **2**:418 (April) 1933.

Children complaining of frequent colds were examined carefully for signs of chronic sinusitis, which were frequently found. The case histories of eighteen patients are presented. A definite connection was seen between chronic nasopharyngitis and chronic sinus infection in children. Marked improvement followed treatment, which was planned to be as simple as possible, depending on the pathologic conditions. The authors state that they are convinced of the value, indeed the necessity, of the proper surgical treatment when pus is present, for "where there is pus, there should be drainage."

LEMASTER, Evanston, Ill. [AM. J. DIS. CHILD.]

METABOLISM OF MINERAL MATTER IN INFLAMMATORY DISEASES OF THE NOSE AND THE NASAL SINUSES. R. MITTERMAIER, Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **134**:147 (Feb. 16) 1933.

Mittermaier studied the mineral content of the dried residue of the mucosae and of their secretions in acute and chronic inflammations of the nose and of the accessory nasal sinuses. The highest increase in mineral content is found in the secretion in acute rhinitis. In chronic inflammation of the maxillary sinus the mineral content remains rather constantly at a high level. An increase in mineral content can be due to accumulation of the secretion in the sinus over a long period. The change of a purulent secretion into a mucoid one is accompanied by an increase in mineral content. It was found that an edematous mucosa contains more minerals than a coarse flat mucosa with inflammatory changes. The increase in fluid content of secretions and mucosae which occurs parallel with the increase in mineral content indicates a tendency to equalize differences in osmotic pressure.

EDITOR'S ABSTRACT.

TREATMENT OF HEADACHE AFTER RHINOLOGIC OPERATIONS, ESPECIALLY SEPTUM RESECTIONS. G. HALÁSZ, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:177 (March 21) 1933.

Halász states that the headaches caused by tamponade after resection of the septum result from a disturbance of the equilibrium of the blood vessels. This hypothesis is supported by the fact that the headaches can be prevented by the administration of substances containing the nitro group, as is the case in migraine. No secondary reactions were observed with this therapy. EDITOR'S ABSTRACT.

A COMPARATIVE ANATOMICOPHYSIOLOGIC STUDY OF THE FUNCTION OF THE ACCESSORY NASAL SINUSES IN HUMAN BEINGS AND OTHER MAMMALS. A. ECKERT-MÖBIUS, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:287 (April 4) 1933.

Eckert-Möbius states that his comparative anatomicophysiologic study of the nasal sinuses of human beings and many other types of mammals confirms the theory that the essential function of the nasal sinuses is to aid in warming and moistening the inspired air, especially in deep respiration. The nasal sinuses, like the laryngeal pouches found in apes, certain species of whales, antelopes and some other animals, increase the surface of the mucosa of the upper respiratory tract; because of their lateral position outside of the main respiratory tract they do this without increasing the so-called dangerous space during respiration. A better perception of smell cannot be brought about by the exchange of air which takes place between the nose and the sinuses even by sniffling. Besides the essential function of the sinuses, their development decreases the weight of the skull and increases the resonance spaces. The extent of the accessory nasal sinuses is determined mainly by the functional demands made on the upper respiratory tract; their distribution and arrangement in the skull, however, are determined chiefly by static-dynamic influences on the shape of the skull.

EDITOR'S ABSTRACT.

SUPPURATION OF THE MAXILLARY SINUS DUE TO TEETH IN THE SINUS. A. BLUMENTHAL, Ztschr. f. Laryng., Rhin. Otol. (Teil 1: Folia oto-laryng.) **24**:62 (March) 1933.

Blumenthal reports a case of suppuration of the maxillary sinus caused by the presence of two teeth in the sinus. The teeth were not supernumerary but were the last two teeth of the left supramaxilla which had developed within the sinus and were missing from the dental arch. At the point of the alveolar process at which the missing teeth should have developed, there was a fistula connecting with the teeth in the sinus. The pus was discharged through this fistula—not through the nose, as is usually the case in suppurations of the maxillary sinus. The location of the two teeth in the posterior wall of the sinus relatively far from the dental arch is not remarkable when one considers that the development of the teeth takes place in a very early period of life in which the lumen of the sinus is small and the individual walls are close together. EDITOR'S ABSTRACT.

INJURIES OF THE ACCESSORY NASAL SINUS. A. L. CSILLAG, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:64 (March) 1933.

In connection with the report of five cases, Csillag gives a comprehensive discussion of injuries of the nasal sinuses, including their complications, symptoms, diagnosis and therapy. If the force causing the trauma is slight only edema or a hematoma may appear in the skin although a fracture of the corresponding sinus wall exists. Stronger forces usually cause injuries of the soft parts covering or adjoining the sinus and many different injuries of the osseous walls of the sinus. Inflammation and suppuration of the sinus mucosa usually follow fracture of the wall, and ulceration and necrosis have also been observed; occasionally the

extravasated blood is resorbed and no inflammatory complication occurs. In injuries of the nasal sinus, injuries to adjacent parts should always be looked for. Meningitis and abscess of the brain are the most dangerous complications. A constant symptom of injury to the sinuses is hemorrhage from the nose or mouth. Ocular symptoms often dominate the clinical picture. Roentgenographic and ophthalmologic examination should always accompany rhinologic examination. Injuries of the maxillary sinus are usually accompanied by other injuries. Isolated injury of the frontal sinus is not infrequent. Injury of the ethmoid sinus usually occurs through the nose or orbit, or by injury to other sinuses. The sphenoid sinus is most rarely involved. The nature and degree of the lesions are best judged by surgical exposure, as the roentgenogram does not show this clearly. The infectious cerebral complications are best avoided by exposure of the seat of the injury, removal of fractured segments of bone and creation of a wide communication with the nasal cavity.

EDITOR'S ABSTRACT.

Miscellaneous

DENTAL FOREIGN BODY IN THE AIR PASSAGES. JULIAN L. ROGATZ, Arch. Pediat. **50**:18 (Jan.) 1933.

The author urges that early roentgenography and bronchoscopy be utilized in the effort to ascertain the presence of a foreign body suspected from the history and physical signs, in order to assure a favorable prognosis in what might otherwise prove to be a fatal case. He calls attention to the danger of aspirated dental material and reports a fatal case in which a child aspirated a molar tooth.

The literature concerning foreign bodies in the air passages is reviewed, and the symptomatology is discussed.

WADDELL, JR., University, Va. [AM. J. DIS. CHILD.]

SOME NEWER NUTRITIONAL FACTS AND THEORIES: THEIR CORRELATION WITH OTOLARYNGOLOGY. J. W. JERVEY, J. South Carolina M. A. **29**:34 (Feb.) 1933.

The writer quotes from several authors such as Osler and Plato, emphasizing the importance of nutrition. It is Jervey's belief that "the disease which flesh is heir to" can in large measure be successfully prevented or controlled by properly enlightened systematic selection of food. The types of disease which can be corrected and frequently cured by proper feeding are diseases due to deficiency, diseases of metabolism, diseases of the blood-forming system, diseases of the circulatory system, diseases of the glands of internal secretion, diseases of the nervous and locomotor systems and all the diseases of the digestive and respiratory systems and of the kidneys not due to specific infections. The writer gives the credit to Jarvis of Vermont for proving that the mucosa covering the anterior portion of the nasal septum can be accepted as an indication of whether the chemical reaction of the body is acid or alkaline. Jervey has found the gingivolabial fold of the lower mandible to be a color indicator of acid or alkaline preponderance. Most of the pathologic micro-organisms attack in an acid medium but have little effect in an alkaline one. There are two ways to control the chemical reaction of the body; (1) the temporary, by therapy with chemicals such as bicarbonate of soda or dilute nitrohydrochloric acid, and (2) the permanent, with proper food. Jervey warns against placing a patient on a "diet," as it is likely to make him a hypochondriac; it is better to tell him that you are selecting better food for him.

The foods are classified as acid and alkaline. To the latter group belong the fresh vegetables, milk and citrus fruits. Animal foods, fish, flesh and fowl, are the acid-producing foods. If these simple facts are kept in mind, it is not difficult to regulate the chemical condition of the body.

WESTON, JR., COLUMBIA, S. C. [AM. J. DIS. CHILD.]

EYE, EAR, NOSE AND THROAT PATHOLOGY IN CHILDREN. A. P. HUDGINS, West Virginia M. J. **29**:160 (April) 1933.

The author makes an appeal for a thorough and complete examination in pathologic conditions of the eye, ear, nose and throat in children, with a search especially for obscure factors. It is not enough to assume, on the discovery of infected tonsils and adenoids, that therein lies the entire explanation of the patient's trouble.

The various pathologic conditions of the eyes, ears, nose and throat in children are discussed fully, together with their relation not only to each other, but to the more general and systemic infections. The importance of early, proper and persistent treatment of the primary foci is urged in order that the more serious secondary conditions may be avoided.

SHAWKEY, Charleston, W. Va. [AM. J. DIS. CHILD.]

THE RESIDUAL SYNDROME OF CLOSED CRANIOCEREBRAL TRAUMATISMS: A STUDY OF FORTY-THREE OBSERVATIONS FROM THE LABYRINTHINE, OPHTHALMOLOGIC AND NEUROPSYCHIATRIC POINTS OF VIEW. J. HELSMOORTEL, JR., L. BAUWENS and L. VAN BOGAERT, Rev. d'oto-neuro-opht. **10**:581 (Sept.-Oct.) 1932.

"There is no problem in neuropsychiatric practice in which the personal equation plays a greater rôle than in the diagnostic and prognostic appreciation of the sequelae of cranial traumatisms." The two current points of view (one that the syndrome is a traumatic neurosis, the other that there is an organic basis for the symptoms) are not irreconcilable. Recent anatomic studies have shown a diffusion of microscopic lesions which involve the nerve tissues, the vessels, the hemato-encephalic barrier and the plexiform apparatus, and which resemble those observed in cases of multiple sclerosis, cerebral arteriosclerosis and epidemic encephalitis. The tissue defect is the organic nucleus around which is crystallized a series of functional disorders and psychogenic reactions.

Study of the forty-three cases, protocols of which are given, is limited to an analysis of: (1) vestibular disturbances, (2) ophthalmodynamometric changes, (3) late neuropsychiatric manifestations and (4) modifications of the cerebrospinal fluid. The vestibular examination must be complete; otherwise, an isolated perturbation may be overlooked. Attention is called to the possibility of the existence of a disturbance of the otolithic apparatus while all other labyrinthine tests yield normal results. The methods devised by Quix are used to determine the state of the otolithic function. Quix believed that alterations in the maculae play the principal rôle in the vertigo that follows cranial trauma. The first vestibular examination was made not earlier than one month after the accident, and the second after several months. Sixteen patients had disturbances of the otolithic apparatus, which was accompanied ten times by hypo-excitability and four times by hyperexcitability of the labyrinth. It is rare to find at the same time hypo-excitability or hyperexcitability to the three tests: caloric, rotatory and galvanic. Spontaneous nystagmus was the sole sign in two patients with concussion of the labyrinth. It was present in seven cases of pure vestibular hypoexcitability, in three cases of pure hyperexcitability and in one case of the mixed form. Spontaneous deviation of the arms did not always accompany the nystagmus. With the galvanic test the average threshold varied between 4 and 6 milliamperes. Patients with the higher thresholds usually had otolithic disturbances also. The Romberg test was positive in only two cases. Spontaneous nystagmus was observed in fourteen patients. The tests of Quix, the galvanic and the caloric, were the fundamentals of the examination, to which were added supplementary tests that were necessary to furnish an exact idea of the state of the vestibular function. The seat of the lesion is usually thought to be in the cerebral *axis*, although Alexander, in one patient, located it in the maculae and cristae. Vertigo is the most persistent symptom and is accompanied by either hyperexcitability or a normal labyrinth. The types of evolution of the disease are classified as follows: 1. The vestibular and otolithic apparatuses may become

normal. 2. The only disturbance persisting may be an otolithic perturbation. 3. A hyperexcitability or a hypo-excitability, found with one test only, may be observed. 4. Isolated hypo-excitability with disturbance of the otolithic apparatus may persist. 5. Isolated hyperexcitability may be found. 6. There may be a mixture of hyperexcitability and hypo-excitability. 7. Hyperexcitability with the otolithic syndrome may be noted. 8. The labyrinth may be inexcitable. Type 3 was observed especially in light degrees of concussion; type 2 is a residual syndrome of light concussion, and types 4 and 5 are residual syndromes of a moderate and grave concussion. Types 5, 6 and 7 are syndromes of transition.

Ophthalmodynamometric observations were made in thirty-five cases. In all cases negative from the otovestibular and neurologic points of view, in four cases with a light syndrome, in one with a marked concussive syndrome and in one negative case the pressure in the central artery of the retina was normal. The pressure was increased in all the other cases, with or without fracture. Hypertension of the central artery of the retina may remain a long time, although, rarely, it may disappear after a number of months. It was found in 69.5 per cent of the established cases examined and only once in a suspected case. True papillary stasis accompanying retinal hypertension was not observed.

In the series of cases the posttraumatic syndrome of dementia, disturbance of olfaction and the focal syndrome were rare. In general, the neurologic semiology was poor, and in a number of cases the results of neurologic examination were negative. Disturbances of tonus were frequent. Pupillary changes were present in four cases and infundibulotuberian disturbances in two. Psychic aberrations are uniformly described by the patient: headache, poor sleep, disequilibration, abnormal sensitiveness to noises, heat and alcohol, giddiness, incapacity for work and distaste for life. When the history varies considerably, a morbid psychopathologic predisposition should be suspected. In most cases the terrain is unfavorable for the development of hysteria, but the continual discussions of the degree of disability cause certain patients to seek claims for damages and to become querulous, which they consciously or unconsciously justify by superadded symptoms. Most often hysteria occurs after slight traumatisms; it develops late, and the absence of neurologic, labyrinthine, ocular and biologic disturbances contrasts with the gravity of the subjective syndrome and permits its recognition.

The pressure of the spinal fluid was raised in sixteen of the twenty-six cases in which it was examined. In forty examinations, the cytologic formula was normal in fourteen; there was hyperalbuminosis in eleven; hyperalbuminosis and a proportional hypercytosis were present in ten, and in five there was hypercytosis alone or out of proportion to the albumin percentage. Four times in seventeen estimations a relatively slight hypoglycorrhachia was observed. The number of cells diminishes rapidly, but the hyperalbuminosis persists for a long time.

In cases with a marked concussive syndrome, the vestibular types 3 and 4 were most often observed. In cases with slight neurologic syndromes, the vestibular syndromes were extremely variable. In cases with a doubtful neurologic syndrome, the appearance of the vestibular types 2 and 4 requires reservation in the diagnosis of traumatic psychosis. When the neurologic examination gave negative results, vestibular and otolithic examinations also gave negative results.

When a slight retinal hypertension existed, there was most often a pure otolithic sequela. With marked retinal hypertension there were apt to be mixed hyperexcitability and hypo-excitability of the labyrinth, representing the beginning of the syndrome of inhibition. Retinal hypertension with a normal vestibular syndrome was observed only once. In two cases with a doubtful neuropsychic syndrome, otolithic disturbances were noted.

The conclusions reached are as follows: 1. There is not necessarily a relation between the gravity of the traumatism and the gravity of the prognosis, immediate or remote. 2. The authors have frequently been surprised at the tenacity of the sequelae observed in their patients. 3. In the important cases it is often difficult to determine the degree of disability. In such cases it is necessary to wait longer than has been the custom, since late complications are not exceptional. 4. Patients

should be examined a number of times, beginning with the surgical stage of the accident and with the surgeon. The family should be questioned early with regard to the heredity and psychologic constitution of the patient. 5. Neuropsychiatric, otovestibular, ocular and biologic examinations should be made and repeated at intervals. 6. Owing to the poverty of objective signs, it is important to consider carefully the advisability of encephalography as a routine method of examination.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

SEVERE INJURIES FOLLOWING ENDOLUMBAR INJECTIONS OF ACRIFLAVINE HYDROCHLORIDE IN RHINOGENOUS AND OTOGENOUS PURULENT MENINGITIS. G. EIGLER and W. GEISLER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:201 (March 21) 1933.

Eigler and Geisler report that in two cases of acute meningitis endolumbar injections of small doses of acriflavine hydrochloride produced severe alterations of the spinal cord which finally resulted in the clinical picture of complete transverse paralysis. The same injuries were observed in experiments on two monkeys. In three other cases of meningitis no improvement was seen after endolumbar injections of acriflavine hydrochloride. It was found that the distribution of the dye following endolumbar injections is limited to the lower subarachnoid spaces. This shows the futility of the therapy in otogenous or rhinogenous meningitis as the medicament does not reach the focus of infection. On the basis of their unfortunate results the authors insistently warn against the use of endolumbar injections of acriflavine hydrochloride. They think that, aside from surgical intervention, the frequent and copious evacuation of the lumbar canal and the intravenous injection of methenamine and acriflavine hydrochloride give the best results in purulent meningitis.

EDITOR'S ABSTRACT.

DIET FOR PATIENTS WITH HAY FEVER. C. E. BENJAMINS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:153 (June 8) 1933.

In an article which appeared in the *Zeitschrift für Hals-, Nasen- und Ohrenheilkunde* (**30**:473 [May 13] 1932; abstr., ARCH. OTOLARYNG. **16**:879 [Dec.] 1932) Benjamins advanced the theory that hay fever may result from the continued use of cereal products. He now suggests various substitutes for cereal flours and outlines a cereal-free diet. He states that good tasty bread can be made without cereal flour, and he gives several recipes utilizing potato and soya bean flour. Good results have been obtained in some cases in which the diet was not commenced until March or April, but the longer it is employed the better is the result, and it is advisable to continue the diet in winter. The author earnestly requests others to report their results with this therapy.

EDITOR'S ABSTRACT.

TUMORS OF THE HYPOPHYSIS AND THE SURROUNDING AREA. J. ERDHEIM, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:1 (March) 1933.

Erdheim states that all tumors of the hypophysis which come into practical consideration involve the anterior lobe. The most important seat of hypophyseal tumors is the sella turcica. Intrasellar tumors, if large enough, cause atrophy of the anterior lobe by pressure, which results in hypophyseal cachexia in adults. Rarely intrasellar tumors are craniopharyngiomas, that is, tumors developed from the epithelial rests of the craniopharyngeal canal which sometimes occur in the portion of the anterior lobe covering the infundibulum and stalk. Such tumors occur in childhood and cause nanism. Eosinophilic adenomas are the more common form of intrasellar tumors. They cause hypersecretion, which results in acromegaly in adults or in gigantism in children. Next in importance as the seat of hypophyseal tumors are the infundibulum and the infundibular stalk. The tumors found there are usually craniopharyngiomas originating in the portion of the anterior lobe

covering the infundibulum and its stalk, but adenomas also occur. Tumors originating in the stalk or growing into it from the sella lead to visual disturbances. Adenomas are usually soft and easily extirpated, whereas craniopharyngiomas may be solid, with a tendency to calcification, or cystic. EDITOR'S ABSTRACT.

A CLINICAL STUDY OF HYPOPHYSEAL TUMORS. B. FLEISCHER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:7 (March) 1933.

Fleischer states that the differential diagnosis of hypophyseal tumors must take into account tumors of the adjoining region, which influence the hypophysis and produce similar symptoms. Of foremost diagnostic importance are the symptoms caused by pressure on the optic chiasm. Bitemporal hemianopia, the classic symptom of hypophyseal tumor, often does not exist in pure form or is incomplete in the early stages. The nature of the beginning and the development of the visual disturbances is particularly important, but the variations in the size and shape of the sella turcica and its position in relation to the chiasm are so great that no far-reaching conclusion may be drawn. Careful perimetry is important because the optic disk is often normal when disturbances of the visual field are extensive. Other important pressure effects are changes at the base of the skull, especially in the sella turcica, which can be demonstrated by stereographic roentgenography. Endocrine disturbances may be the only symptoms of a hypophyseal tumor if its size or location does not cause pressure symptoms. In adenomas, which are the most common growths, according to Cushing, the bitemporal visual defects may be of various types or may be lacking, and there is simple optic atrophy. These growths are usually of intrasellar origin, and the distention of the floor of the sella toward the sphenoid sinus and of the wall anteriorly and posteriorly is a characteristic roentgenologic sign. They may cause hyperpituitarism (gigantism or acromegaly) or more often hypopituitarism. They occur at various ages, but are most common in middle age. Craniopharyngiomas (tumors developed from embryonic rests of the craniopharyngeal canal) cause bitemporal and, occasionally, central visual defects. Besides simple optic atrophy they may cause choked disk. They are suprasellar. They usually occur in youth, causing infantilism; if they occur later they produce hypopituitarism with adiposogenital dystrophy. When advanced they cause symptoms of the tuber cinereum, polyuria, lethargy and adiposity. Roentgenologically, there is a shallow widening of the sella, and frequently shadows of calcium concretions may be seen. Chiasmal symptoms similar to those produced by hypophyseal tumors are produced by meningiomas developing in the region of the sulcus of the chiasm. The hemianopia is frequently unilateral at first but later becomes bitemporal; there is simple optic atrophy. The tumors are suprasellar; the sella is normal. Growth is slow, and endocrine symptoms appear late. The growths occur chiefly in middle age. Gliomas of the chiasm predominate in childhood. They cause visual disturbances, usually not typically hemianoptic, and simple optic atrophy. The tumors are of suprasellar origin and cause a pear-shaped anterior widening of the sella and the optic foramen. The hypophyseal symptoms are insignificant; symptoms of neurofibromatosis may exist. Aneurysms should also be considered in differential diagnosis. EDITOR'S ABSTRACT.

SURGICAL THERAPY OF TUMORS OF THE HYPOPHYSIS WITH SPECIAL CONSIDERATION OF ENDOCRANIAL PROCEDURE. O. KLEINSCHMIDT, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:28 (March) 1933.

Kleinschmidt states that the efficacy of roentgen therapy is limited to certain types of hypophyseal tumors, chiefly adenomas. Primary roentgen irradiation is therefore not justified in all cases, but irradiation after decompression by trepanation or extirpation is valuable. Surgical intervention is indicated when typical visual disturbances occur as a result of compression of the chiasm or optic nerves. The rapid progress of a malignant tumor also demands intervention, perhaps only

trepanation to relieve pressure; this rule also applies to large cysts. Endocrine disturbances alone are not sufficient indication. Two methods come into consideration: the transphenoidal and the intracranial. The first is more simple but there is danger of meningitis and a fistula; moreover, if the tumor has extended beyond the bed of the hypophysis above, radical extirpation is impossible. The second method is more radical and permits operation in the suprasellar region under visual control, but a radical extirpation of the hypophyseal bed is not certain. The transphenoidal method is considered better for intrasellar tumors, and the intracranial method for suprasellar tumors; concerning craniosellar tumors, opinion is divided. The author discusses briefly some of the transphenoidal methods. In the intracranial methods a flap is made in the frontal region, opening of the frontal sinus being avoided, if possible. Then, by Krause and Cushing's method, the dura with the frontal lobe is detached from the orbital roof and is incised at the region of the sella. Intervention is usually unilateral except in case of severe hemorrhage. Pressure is relieved by the injection of hypertonic salt solution or by puncture of the ventricle. After careful enucleation of the tumor, the capsule should be enucleated. The mortality has been greatly reduced by this procedure. Relief of headaches and visual disturbances is obtained; in this respect the intracranial method is better as it permits removal of the capsule and relief of the pressure caused by it. It is too early to give statistics on the end-results of this method, but as it is more radical it is to be expected that there will be fewer recurrences than with other methods.

EDITOR'S ABSTRACT.

BLOOD DYSCRASIAS AFTER INFECTION OF THE EAR, NOSE OR THROAT. W. KINDLER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:39 (March) 1933.

Kindler reviews an earlier observation of a case of acute myeloid leukemia which occurred immediately after a radical operation on the maxillary sinus in a previously healthy man. He also reports three new cases of dyscrasias of the blood occurring after infection of the otic or tonsillar regions. The first of these was a case of acute fatal leukemia following suppurative perichondritis of the auricle. The second case was one of a subacute leukemia with an aleukemic prodromal stage which followed nonspecific inflammation of the tonsils and cervical glands. The third case was one of purpura haemorrhagica with spontaneous, severe, late hemorrhage from the tonsillar abscess cavity, without any other manifest or latent signs of a hemorrhagic diathesis. A blood platelet count showed a severe thrombopenia as the cause of hemorrhage. An increase in the number of blood platelets and disappearance of hemorrhages followed injections of congo red. In the presence of a hemorrhagic diathesis acute and chronic infections of the tonsils play an important part in determining the occurrence and localization of hemorrhages in the pharyngopalatine region.

EDITOR'S ABSTRACT.

CONGENITAL ESOPHAGEAL STENOSIS. STEN ECKERSTRÖM, Acta pædiat. **13**:63 (June 6) 1932.

Esophageal stenosis, which is more rare than atresia, may permit survival; however, there is always limitation of growth, and in certain reported cases there has been retardation of sexual maturity.

Eckerström reports the case of a boy, aged 8 years, who had difficulty in swallowing and who at the time of examination was 12 per cent underheight and 44 per cent underweight for his age. Roentgenograms showed a constriction of the esophagus at about the level of the eighth thoracic vertebra, with great dilation above it; a fine stream of the opaque meal passed the stricture, but the upper part was not emptied even in half an hour.

With the esophagoscope a diaphragm was seen at the level indicated, showing a small eccentric aperture. This was enlarged with the galvanocautery so as to

admit a no. 18 stomach tube, which was left in place for a week. Within the next ten days the opening was dilated to the size of a no. 40 sound. From this time on the patient was able to eat a normal diet without any untoward symptoms. In three months he gained 4.5 Kg. in weight and 2 cm. in height.

McIntosh, New York. [Am. J. Dis. Child.]

Congenital Stenosis of the Esophagus. Leonard Findlay, Acta pædiat. **13**:70 (June 6) 1932.

In an effort to elucidate the etiology and pathology of congenital esophageal stricture, Findlay has reviewed the eighty cases reported as occurring in children under 15 years of age and has selected for analysis twenty-three of these, in which the nature of the abnormality was demonstrated by autopsy, operation or esophagoscopy. Four types of lesion were identified: (1) narrowing of the lumen (nine cases), (2) membranous diaphragm (four cases), (3) fibrous or fibromuscular thickening of the wall (eight cases) and (4) spasm (two cases). The distribution of these different sorts of obstruction along the course of the esophagus was so varied as to throw doubt on the truth of the familiar clinical aphorism that pathologic narrowings are likely to occur either at the upper or lower end of the tube or at the level of the bifurcation of the trachea. The same evidence argues against stenosis as being a mild degree of congenital atresia, since the latter always occurs at the bifurcation (from the fourth to the sixth thoracic vertebral body). Simple narrowing and the formation of a membrane represent ontogenetic anomalies. Regarding the origin of the other two types of obstruction, no general statement can at present be made.

McIntosh, New York. [Am. J. Dis. Child.]

Society Transactions

MASSACHUSETTS EYE AND EAR INFIRMARY, OTO-RHINO-LARYNGOLOGICAL SERVICES

Regular Clinical Meetings, September, 1932

EDITED BY PHILIP E. MELTZER, M.D.

RHINOSCLEROMA. DR. PHILIP MYSEL.

Before presenting the patient, I should like to say a few words about the disease. Rhinoscleroma was first described by Hebra in 1870. It is a rare, chronic, incurable disease, possibly contagious and occurring only in certain districts of the world, namely, Russia, Galacea, Roumania and Prussia. It has lately invaded Hungary and is gaining a foothold in Germany, Italy and Switzerland. All the cases seen in this country are imported.

The disease usually starts as a dense, hard plaque at the mucocutaneous border within the nostril. After inception, the disease spreads by a slow but irresistible progress either by enlargement of the original plaques or by new centers of development. This progressive inflammation rapidly develops cicatricial tissue and produces visible changes in the nose, nasopharynx and larynx. It may be limited to the nose, nasopharynx, larynx or trachea, but it seldom invades the bronchi. Characteristic of the disease is the lack of inflammatory reaction; there is no tendency to ulcerate, but there are generally cicatrization and contraction into scar tissue of cartilage-like hardness. Adhesions result in the form of semilunar bands, diaphragms and strictures.

The disease is caused by the Bacillus of Frisch, related to the Bacillus of Friedländer. The organism is gram-negative, is found in pure cultures and is present with hyaline bodies and a large cell with a homogeneous protoplasm which stains poorly—the so-called fat droplet cell of Mikulicz.

When the nose is involved, the symptoms are those of a painless and slowly increasing obstruction and catarrh, progressing to complete stenosis. If the larynx is affected, the disease causes catarrh, cough, hoarseness and, finally, dyspnea and stenosis. This sequence may spread over fifteen, twenty or more years, and the prodromal catarrh may persist for years before the formation of scleromatous infiltration.

As to the prognosis, the prospect for a complete cure is poor. The general health may not be impaired unless stenosis becomes marked. The disease progresses slowly or remains quiescent for years and then becomes active.

The treatment has been unsatisfactory. Roentgen rays and radium have been used without important results. Treatment is mostly symptomatic. The obstructing infiltrations are removed, and the diseased area is kept as clean as possible. Tracheotomy may be necessary. Thyrotomy may be performed, followed by dilatation. Drugs are useless.

This patient is a Russian housewife, aged 43. Her chief complaint is intermittent attacks of hoarseness for the past seven years. Lately the patient has had a cough with expectoration, but there is no blood in the sputum. On examination, she showed a hard plaque on the right inferior turbinate, but no redness or ulceration. Posterior rhinoscopy showed that the choanae are narrowed, especially on the right, with nodular infiltration but no pus or polypi. The larynx shows a subglottic infiltration. The usual subglottic concavity and tracheal rings cannot be made out. The arytenoids and aryepiglottic folds are also involved in a nodular infiltration. There is no sign of inflammation, and there is no tendency

to ulcerate or break down. A biopsy specimen taken from the larynx showed the Frisch bacillus and the characteristic cells of Mikulicz.

A microscopic examination was made by Dr. Delbert Judd. A slide made of a section taken from the arytenoid was shown. The microscopic characteristics of the disease as shown in this section are a dense infiltration of the stroma with plasma cells, histiocytes and reticulo-endothelial cells. The latter cells become enlarged by the formation of vacuoles within the cytoplasm, and the nucleus becomes shrunken and disappears in many of the cells. When the cells take on the characteristics of being able to disappear, they become known as Mikulicz cells. A large number of them in the stroma gives it a peculiar frothy appearance. Hyaline bodies are frequently formed within the cells. The bacilli accumulate in large numbers within the Mikulicz cells.

Frontal Sinusitis with Osteomyelitis and Abscess of the Frontal Lobe. Dr. R. L. Goodale.

I first saw the patient four years ago; he had pansinusitis and complained of fatigue and headache which was generalized over the forehead. At that time he was a student at Massachusetts Institute of Technology and did not want to spend much time in the hospital, so I performed a bilateral Caldwell-Luc operation and anterior ethmoidectomy in order to give him aeration and drainage of the frontal sinuses. Until this winter he improved slowly; this past winter, however, he had increased right frontal headache and fatigue. A roentgenogram showed no change, merely pansinusitis with some reaction of the bone around the frontal sinus, but no osteomyelitis. On June 21, 1932, I removed the floor of the frontal sinuses and made large drainage openings into the nose. At that time I removed some of the polypoid tissue from the ethmoidal region. Immediately after operation marked edema developed over both frontal sinuses, extending up toward the hairline. The edema lasted four days and gradually localized to the root of the nose and to the adjacent side of the right frontal sinus. There was persistent marked tenderness over this region. I reopened the operative wound, thinking that there might be a collection of pus under the soft tissues, but none was found. Drains were inserted. Both sides drained for a considerable time, and a roentgenogram taken on July 27 showed beginning osteomyelitis of the anterior wall of both frontal sinuses. I opened both sinuses, performed an obliterative operation and left the wound wide open, with an incision to the hairline and from one eyebrow to the other. Secondary anemia developed, and the patient was given two transfusions during the course of the next month. Repeated roentgenograms were taken to discover further extension of the osteomyelitis. The last roentgenogram showed extension of the osteomyelitis into the external angle of the right frontal sinus and also into the internal angle. Dr. H. P. Mosher and Dr. D. Campbell Smyth were called in consultation. The patient began to complain of pain in the right eye, and he seemed to have considerable headache which was localized on the right side but was not definite. It would come and go but was worse after each dressing. I planned to take off the frontal bone and uncover the dura the day after the consultation; however, to my surprise, the patient died suddenly before this could be done. Evidently death was due to sudden rupture of an abscess of the brain into the ventricle. The problem I should like to solve is how I could have discovered the abscess during the past few months, in view of the facts that the neurologic examination and the examination of the eyes gave negative results, and that there was no localized sign other than headache which occurred mostly after the dressing.

DISCUSSION

Dr. H. P. Mosher: This case is courageously reported. It is pathetic. I do not know what to say in answer to Dr. Goodale's question as to whether the abscess could have been discovered earlier except that one should always suspect the possible presence of an abscess of the brain with osteomyelitis. In my experience the Lynch operation will close up the inner angle. I believe that osteomyelitis

is due to the virulence of the organism rather than to the trauma of the frontal wall. The patient in all probability had osteomyelitis when he had edema of the scalp, and osteomyelitis was present from then till death. I believe that edema of the forehead is a very positive sign that the medulla of the bone is infected to the limit of the edema. In this case the osteomyelitis probably subsided and flared up later. As to the procedure, when Dr. Smyth and I saw the patient in consultation, we agreed to open the frontal bone all the way through to the dura to see whether or not it was actively diseased; an extradural abscess, if present, would then have been discovered. I believe that an exploratory operation on the skull is an advance in the treatment of osteomyelitis. Dr. Goodale also asked whether there was any hurry about the operation. It was getting along toward the end of the week and I said, "No, two or three days will not make much difference." The man was to have been operated on on Tuesday, and on Monday he died. I think that early trephining of the frontal bone in all these cases is indicated, anywhere except over the longitudinal sinus.

DR. M. G. EVANS: Does Dr. Mosher mean to trephine the skull at the first sign of osteomyelitis?

DR. H. P. MOSHER: Yes, to the limit of the edema.

DR. D. C. SMYTH: At autopsy the patient showed no meningitis. The dura appeared normal, and was in immediate contact with the anterior table of the frontal bone. Even if the dura had been exposed at the proposed operation, I doubt whether there was enough indication from its appearance to warrant exploration of the brain. Decompression might have given the patient relief and postponed death.

FIBROMA OF THE NASOPHARYNX. DR. F. L. WEILLE.

The patient, aged 14 years, was admitted to the hospital complaining of a left nasal obstruction of two years' duration. Examination showed a large nasopharyngeal tumor pushing the soft palate forward. Dr. E. W. Herman advised partial removal of the tumor through the mouth and pharynx and instructed me, as I performed the operation under his direction. The soft palate was resected so as to expose the growth. A tonsil wire was inserted and firmly tightened around the growth so as to remove about two-thirds, leaving a stump of about one-third. After the snare had been placed, six 1 mm. gold seeds were placed in the stump of the tumor. Then the major portion was removed with diathermy, and the stump was left intact as a mold for the gold seeds. Not more than 1 cc. of blood was lost during the entire procedure, which was practically bloodless. After the loop was drawn home in the tonsil snare, the diathermy simply cooked the stump. It was such a relatively easy piece of surgery I was much impressed. The patient had an uneventful and bloodless convalescence.

DISCUSSION

DR. H. P. MOSHER: Do I understand that the seeds were put in after the snare was adjusted? Was there any question whether the seeds were going to be squeezed out?

DR. E. W. HERMAN: They were placed 1 cm. away. I believe that the handling of nasopharyngeal fibromas is rather simple so long as they do not metastasize and so long as there is no danger of the patient's dying. In the second place, they are always almost attached to the base of the sphenoid sinus, and you cannot remove them surgically in toto. They are radiosensitive. If the six seeds are not sufficient to destroy them—if a nodule remains a year or six months from now—two more seeds can be put in, and eventually satisfactory results will be obtained. I treated a number of patients with the snare and diathermy combined; by going slowly one can control the bleeding in the major portion. Of course, if the bleeding extended extensively, one would have to change the procedure a little. In the usual case I think that the method used by Dr. Weille is the more satisfactory one for surgically removing the growth.

CYST OF THE BRANCHIAL CLEFT SIMULATING PERITONSILLAR ABSCESS. DR. F. L. WEILLE.

The patient, aged 25, was seen by me ten days ago with the chief complaint of sore throat and difficulty in swallowing, with swelling of the right side of the neck. Examination showed a reddened tonsil on that side, which projected slightly toward the midline. There were injection of the pillars, no exudate and some edema of the neck, with a mass at the angle of the jaw which was firm and tender. The mass did not feel fluctuant, and I could not discern what it was. The patient gave a history of having had a swelling four years before in the angle of the jaw; this occurred whenever he ate. It would come and go. The patient has never had anything similar to the present attack. He was treated with hot applications inside and with ice on the outside, and in about eighteen hours pus in considerable amount was expressed from the anterior angle of the tonsil. With pressure on the neck, further pus was expressed through the anterior tonsillar pillar. The patient was relieved for about two days, when the swelling in the neck recurred. I put a probe in the opening in the pillar and thin, serous fluid came out. This continued for several days in small amount. When the patient was discharged, there was a small mass in the neck with a fistula toward the anterior tonsillar pillar. Before discharging him, I placed iodized poppy seed oil 40 per cent in the fissure, and the picture of an annular stricture extending toward the angle of the jaw was obtained. My impression is that the patient had an infection of the tonsil and an infected cyst of the branchial cleft which probably contained salivary gland tissue.

DISCUSSION

DR. E. W. HERMAN: Would the fissure not open behind, rather than in front of, the tonsil?

DR. H. P. MOSHER: I do not know whether or not that is a cleft. The second cleft empties by way of the tonsil, but the common feature is an infection of the submaxillary gland. In other words, I should like to ask Dr. Weille whether it is not the ordinary infection of the submaxillary gland with pus in the neck. The gland is a little too low to be a parotid gland. The fact that pus came out through the anterior pillar is in line with the gland's being a submaxillary gland.

DR. F. L. WEILLE: In the first place, I tried to pass a probe through Whortman's duct and could not do so, and I tried to see whether there was a stone in the submaxillary gland, but there was none.

DR. H. P. MOSHER: That is in favor of a cyst.

DR. F. L. WEILLE: I removed all the tissue from the tonsils and by microscopic examination found it to be that of a salivary gland. I understand from the literature that such tissue can be present in the tonsil.

PAROTID FISTULA. DR. G. H. TOBEY.

A young man underwent a simple operation on the mastoid about ten years ago. For ten years, at intervals, he came to the outpatient department complaining of a discharge from behind the ear. The physicians always told him that there was no fistula or hole there. On the morning I saw him, you could not see one. He said that if he could chew an apple, he could start the discharge. We gave him an apple, and one could see the fluid almost squirt out of the lower end of the mastoid wound and run down the neck; the lesion was evidently a parotid fistula. The patient was admitted to the hospital. Just in front of the tip of the mastoid a probe entered for one-half inch (1.27 cm.) in a forward direction. In order to find the opening, I operated under local anesthesia. With a lacrimal dilator I was able to dilate the fistula much better. The duct was dissected out. Unfortunately, the probe perforated the duct close to the parotid gland. I put a purse-string suture around the gland, including the tissue over it. Having destroyed the tissue with diathermy, I tightened the purse-string suture. At present, the fistula is closed. There was little space in which to work, and I had to be careful

of the facial nerve. During the operation, in order to be sure that I was working along the right tract, I had the patient suck a lemon; throughout the procedure the fluid was injected through the fistula until coagulation occurred and the sutures were applied.

STRICTURE OF THE ESOPHAGUS. DR. H. P. MOSHER.

The question as to how safe it is to pass the esophagoscope to find the amount of stricture when a patient has swallowed a corrosive presents itself to my mind. I have been debating in a number of cases as to how soon to pass the esophagoscope. I remember one case vividly in which Dr. H. Barnes passed it at the end of two weeks. The result was death. The esophagoscope became lodged in the trachea, and the patient died of mediastinitis. A short while ago, Dr. H. Tobey had a patient who had swallowed ammonia. When she entered the hospital she was able to swallow liquids. The question was when it would be safe to look down the esophagus. I suggested a delay of about six weeks. That was pure guesswork. The roentgenogram showed a slight stricture behind the cricoid cartilage, one behind the arch of the aorta and one at the terminal portion of the esophagus, which is where one expects the corrosive to act most violently. The patient was examined later. It was found that she had a very narrow stricture at the terminal portion of the esophagus. It was in this case that I had an opportunity to reexamine the patient at the end of six weeks. I found that the mucous membrane is boggy, but entirely healed. There is no erosion from the corrosive. At the lower end of the esophagus there is a pinpoint stricture, showing that at the end of six weeks the esophagus was slowly occluding. The esophagus can be dilated with a no. 22 esophagoscope; this indicates that the patient is out of the emergency class.

DISCUSSION

DR. H. TOBEY: I think that last year or the year before there was another patient who was examined at the end of five weeks; the upper end of the esophagus looked edematous, and I wondered whether the character of the fluid swallowed had anything to do with it. I think that the corrosive was lye.

DR. C. T. PORTER: I think that the character of the fluid swallowed makes a difference. Was there bleeding in this case?

DR. H. TOBEY: No; however, some of the tissues did not look too firm.

DR. D. C. SMYTH: Are you going to try to pass bougies without an anesthetic?

DR. H. TOBEY: No; not for a white. The opening is so small that anesthesia would be necessary.

DR. C. T. PORTER: I had a case of stricture due to lye on my last service; it was about 3 or 4 inches (8 or 10 cm.) below the cricoid cartilage. I passed the esophagoscope at the end of two or three weeks and could not see the opening.

DR. D. C. SMYTH: The patient remained on my service when you left. At present the esophagus can be dilated with a no. 32 esophagoscope, with retrograde bouginage. She is eating soft solids. The opening was a pinpoint when I saw it. In spite of the fact that in two months a no. 32 esophagoscope can be passed, I think that I should continue the gastrostomy a year or so longer, so that the esophagus can be examined from above.

DERMOID CYST OF THE NECK. DR. E. N. WRIGHT.

A girl, aged 16, entered the hospital with a swelling about the size of a large grape just above the thyroid gland. It has been there as long as she can remember. She gives a history of raising about 1 tablespoonful of blood every two or three weeks. She says that at first the growth got smaller after she raised the blood. In general, there has been little change in the size of the growth. Dr. Harold Tobey operated on the patient.

DISCUSSION

DR. HAROLD TOBEY: At operation a tumor was easily exposed. It seemed to have a thin capsule, with a very thin pedicle, and it did not appear to extend far. I split the hyoid bone, shelled out the cyst and found that the pedicle extended to the floor of the mouth. Incidentally, there was no pathologic condition of the base of the tongue. The cyst was opened in the laboratory, and hair was found in it.

CARCINOMA. DR. PERCY IRELAND and DR. H. P. MOSHER.

The following cases of carcinoma, determined according to biopsy, were seen in Dr. Mosher's laboratory since it opened in 1928.

Location in order of frequency	Cases
Larynx, including all the lesions primary in the larynx	86
Esophagus	50
Ear, including the canal, the middle ear and the mastoid	30
Sinuses, including all the accessory sinuses	28
Pharynx, including the pyriform sinus, the postcricoid and the epiglottis	23
Tonsil (plus 4 anterior pillars)	21
Palate, including the soft and hard palate, unless the antrum is involved	19
Tongue	13
Nasopharynx	6
Miscellaneous, including the turbinate bone, septum, vestibule, mandible, maxilla, orbit, skin, bronchus, thyroid, etc	82
Total	362
Average cases a year	100

COLLEGE OF PHYSICIANS OF PHILADELPHIA AND NEW YORK ACADEMY OF MEDICINE, SECTIONS OF OTOLARYNGOLOGY

Joint Meeting, Nov. 16, 1932

BENJAMIN PARISH, M.D., *Chairman*

POSTOPERATIVE PULMONARY COMPLICATIONS; ARE THEY PREVENTABLE? DR. CHEVALIER JACKSON, Philadelphia.

This subject may be considered best by presenting clinical cases arranged in three groups: (1) cases of postoperative atelectasis; (2) cases of abscess following tonsillectomy, and (3) cases of inspiration of instruments or of other substances at the time of operation. These three groups are distinct. Postoperative atelectasis follows abdominal and occasionally pelvic operations; it is not primarily suppurative, and one does not see it after tonsillectomy or other operations about the head or the upper air passages. In group 2, on the contrary, a sudden onset with fulminating symptoms of abscess, including foul expectoration, follows immediately or shortly after tonsillectomy or an operation about the upper air passages. This type of suppuration does not follow abdominal or pelvic operations unless it is due to a septic infarct. In group 3 there is the long symptomless interval characteristic of the inspiration of a foreign body. In ten cases portions of instruments used in operation on the tonsils have been removed; in sixty cases, teeth dislodged at operation, and in one case, a tonsil. There was a symptomless interval in all of these cases, and no suppuration was present except in the cases in which the foreign body had been present in the lung for a long time, months rather than days. In the case in which a tonsil was found, the tonsillar tissue, presumably containing all infections usually present in diseased lymphoid tissue, was in the bronchus for seven months without producing an abscess; for the first three

months there were no symptoms. It is obvious that the whole question of postoperative suppuration is closely related to the whole problem of infections descending from the upper air passages to the lungs. Until the route of infection and the other etiologic factors have been determined, it will be impossible to answer the question as to whether or not these complications are preventable. The route by the air passages is direct, as is proved by the foreign bodies lost at operation; the route by the blood vessels is equally direct, as was shown by Fetterolf and Fox. The route by the lymph channels may be from the field of operation in the upper air passages or from the tracheobronchial tree by way of the local lymphatics in the lung, though many questions in regard to the latter group of lymphatics are as yet unanswered. There can be no doubt that, so far as is possible, blood should be prevented from running down into the tracheobronchial tree. This blood not only is infected but furnishes a culture medium; it runs down into the tracheobronchial tree at practically every operation, and yet pulmonary abscess following tonsillectomy is exceedingly rare as compared to the number of tonsillectomies. One thing of importance in connection with the inspiration of blood is that the cricopharyngeal pinch-cock maintains the esophagus constantly closed except during swallowing. The larynx, on the contrary, is constantly open; it is located at the bottom of a funnel formed by the pharynx, and into this funnel blood pours from the wounds in the tonsils and nasopharynx. Some blood is swallowed, but the swallowing is intermittent, whereas the flow of blood into the larynx is constant.

DISCUSSION

Dr. J. D. Kernan, New York: Prevention of these complications is what the otolaryngologist is attempting to accomplish. Prophylaxis involves removal of foci in the upper air passages, such as those in diseased sinuses and infected tonsils and teeth. Would it not be possible to block the flow of blood into the trachea by giving anesthesia through the Flag tracheal tube, and why could one not clean the lungs after operation by using intratracheal suction as a routine measure?

Dr. Chevalier Jackson, Philadelphia: One need not tie oneself to the question of blood going down the trachea. Various devices to catch the blood and particles, such as Dr. Moore's tonsillectomy sump, have been invented. The transmission of infection through the blood vessels and lymphatics must also be taken into consideration.

Atresia of the Choanae: Its Incidence, Types and Cause. Dr. J. Parsons Schaeffer, Philadelphia.

Atresia of the choanae or of the pharynx may be congenital or acquired, partial or complete. Acquired atresias are more apt to be pharyngeal, and congenital atresias, choanal. The acquired atresias are due to a number of causes: Syphilitic ulceration accounts for many cases; surgical trauma and other traumas appear to be the determining factors in some cases; then follow, in order, diphtheria, tuberculosis and other simple inflammations. Many of the acquired atresias are, however, located not at the choanae but lower in the pharynx; this results in an occlusion between the nasal pharynx and the oral pharynx.

This paper is limited to a consideration of congenital atresia of the true choanae. The occlusion may be absolute or partial; the atresic mass, epithelial, membranous, osseous or a combination of these. While the condition appears to be relatively uncommon, its occurrence is doubtless more frequent than is generally believed. In all likelihood some atresias of the posterior nares pass unobserved in stillborn infants and in infants that die shortly after birth.

In the living infant with absolute occlusion of the choanae, signs suggestive of nasal obstruction are manifested early. When the occlusion is partial, even though the choanal apertures are small, it may not be recognized for some time. Indeed, some persons reach adult life with partial atresia of the choanae, which in various ways hinders the intake of air by way of the nose. Spontaneous rupture of the

atresic mass, especially if the mass is thin and membranous, may occur early in infancy. As a rule, however, operative measures are required to relieve the obstruction.

The explanation of congenital atresia of the true definitive choanae or posterior nares must be sought primarily (1) in the nasal fossae and the early oral cavity and (2) in the behavior of the floor of the early nasal fossae dorsal to the primitive choanae.

Should the primitive choanae just dorsal to the primitive palate not form during the process of development, the epithelial lining of the primitive nasal fossae and the epithelial lining of the primitive oral cavity would remain intact, each separated from the other by the thinned mesenchymal floor of the early nasal fossae. The continued dorsal growth of the nasal fossae into the mesenchymal mass of the nasofrontal process would ultimately, in cases of maximal growth, lead to a condition in which the epithelium of the nasal fossae would come in contact with the epithelium of the foregut, i. e., the nasal portion of the pharynx, with a variable amount of mesenchymal tissue between the epithelial surfaces. This would result in blindly ending, definitive nasal fossae dorsally; in other words, atresia of the choanae of the posterior nares. If the amount of mesenchyme between the pharyngeal and the nasal epithelium should remain thick, an osseous atresia would ultimately be formed. The atresia of the choanae would, therefore, be membranous, osseous or a combination of these, depending on the degree of dorsal growth of the nasal fossae and the extent of the resorption of the mesenchyme between the nasal and the pharyngeal epithelium.

Even though the primitive choanae were established normally in the roof of the mouth, should the floor of the primitive nasal fossae dorsal to the primitive choanae not be resorbed, there could be no connection with the nasopharynx, save by way of the mouth and the oral pharynx.

The palatal processes developing beneath the floor of the early nasal fossae and fusing with each other in the midline in the formation of the secondary palate would come into intimate relationship with the persistent primitive floor of the nasal fossae dorsal to the primitive choanae and with the nasal floor when the primitive choanae were not established. Fusion between the palatal processes and the primitive nasal floor would in time take place.

DISCUSSION

DR. E. R. FAULKNER, New York: I believe that it is possible that a large number of babies and children who die of respiratory difficulty have a congenital condition similar to atresia of the choanae. One should determine the patency of the choanae before operation rather than suppose that adenoids are responsible for obstruction.

RECENT DEVELOPMENTS IN REGARD TO OTOSCLEROSIS, CHOLESTEATOMA AND THE STATIC LABYRINTH. DR. GEORGE M. COATES, Philadelphia.

As the title suggests, this paper is a résumé of certain phases of the work of several distinguished investigators — work that should be of interest to every otologist.

Otosclerosis.—This section is based on the work of Polvogt and Guild in Baltimore. The usual pathologic process of otosclerosis, as accepted by otologists, is confined to the area surrounding the oval window, causing ossification of the annular ligament with fixation of the foot plate of the stapes in the oval window. Polvogt and Guild had the opportunity of studying by serial section many temporal bones of patients for whom audiometric records had been made. In some of these bones they discovered definite areas of otosclerosis which had not caused the usually accepted symptoms and signs and which in some instances were not in the usual locations. Guild reported eight cases, and Polvogt added two, in which the familial and personal history and the clinical symptoms did not suggest otosclerosis but definite otosclerotic areas were found on sectioning. Some of these areas were in

a very early stage, some were active, and others were entirely quiescent, although evidently of long duration; this partially confirms the clinical fact that the disease is at times self-limited and is not uniformly progressive.

Because of the lack of clinical symptoms, many cases were classed as "early," although they may have been old pathologically. The changes may begin as early as the first year of life; they do not always appear first near the anterior margin of the oval window, nor is the fissula ante fenestram always involved.

In early otosclerosis about the round window, when the condition reaches the stage of ankylosis, one can usually diagnose the uncomplicated cases. In otosclerosis of other regions, affecting the cochlea and producing the nerve type of deafness, Nager, of Zurich, showed that the condition cannot be distinguished clinically from manifestations produced by other causes.

Cholesteatoma.—On the basis of Wittmaac's theories of pneumatization of the temporal bone and some practical applications worked out in Kopetzky's clinic, Almour advocates the utilization of the cholesteatomatous matrix for the epidermization of the mastoid cavity after a radical operation on the mastoid. The normally pneumatized mastoid process, it is now believed, is formed by invasion and replacement of the bone marrow by embryonal myxomatous connective tissue, which fills the middle ear and antrum at birth. This tissue with its limiting epithelium is drawn into the intertrabecular spaces and by contraction eventually lines them, thus affording a more or less direct communication with the antrum and the middle ear. Any interference with this normal development produces either a diploic or a sclerotic mastoid—in other words, an undeveloped mastoid process, or possibly a mixed type, depending on the stage of development at which the interference occurred. Interference may be by an uninfected foreign body, such as meconium or vomitus, or by a microbic infection taking place before development. In either instance a fibrosis is induced which interferes with the ingrowth of the subepithelial tissue into the mastoid process. Thus a sclerotic mastoid process is not viewed as pathologic in itself or as the result of a chronic infection in the middle ear or antrum.

When it can be proved by roentgen examination that the mastoid process is nonpneumatic and wholly sclerotic, Kopetzky and Almour confine the radical operation on the mastoid to the removal of the thick cortex overlying the antrum, without any attempt to excavate the remainder of the process, which is considered healthy bone. The cholesteatomatous mass is removed with care from the uncovered antrum, the posterior wall of the canal reduced and the middle ear cavity treated in the usual manner. This procedure materially shortens the time of operation and leaves a much smaller area to be epidermized. It is considered necessary, in making the plastic flap, that the meatal opening be ample, in order to provide adequate escape for epithelial débris, since otherwise cholesteatoma may redevelop. In other words, cholesteatoma is an attempt at a reparative process, which may succeed perfectly if the avenue of escape for exfoliated epithelial débris is adequate. Therefore, these observers advocate the retention of the cholesteatomatous matrix, which is nothing more than an epidermal covering similar to the one which the operator hopes will eventually line the cavity after the radical operation on the mastoid. It is now believed that the old teaching, following the observation of Kirchner, is unnecessary, the cause of recurrence of the cholesteatomatous mass being an inadequate outlet.

The Equilibrial Mechanism of the Frog.—McNally and Tait have reached definite conclusions which seem logical and clarifying as to the function of certain portions of the static labyrinth, at least in the frog. They believe that the saccule (with its macule and otoliths) has no function in the production of equilibration, but appears to be a rudimentary part of the auditory system.

Response to angular acceleration in rotation (slow speed) is a function of the semicircular canals, whereas response to centrifugal force (rapid turning) is otolithic stimulation in the utricle. The response to gravity gained by tilting the table is similar to the latter reaction, as it is abolished by cutting the nerve to the utricle.

Static function arises from the semicircular canals, while the otoliths serve as a kinetic mechanism. If both labyrinths are removed from a frog, it may assume normal or unusual positions, but it maintains its position in spite of the lack of labyrinths; therefore, the position is due to a neuromuscular mechanism.

When a normal frog is placed on a plane and the plane is tipped slowly so that the movement is below the threshold of stimulation of the canals, the frog responds by discontinuous shifts. If the utricle were steadily stimulated, the shift would be steady and not discontinuous.

If one labyrinth is removed from a frog, the animal leans to one side. When such a frog is manipulated and its posture brought to the midline, the frog maintains its new symmetrical position when released; the moment the head is jarred or moved, however, the animal regains the asymmetrical position. In a decerebrated frog labyrinthine movements are normal, but voluntary movements are curtailed. It is the necessary jar of the utricular otolith that causes response. There is no steady pressure, but there is a sudden stimulus.

When all six canals are removed from a frog, only the utricles remain as active vestibular end-organs. The posture is normal at rest, but any spontaneous movement causes the animal to sway, and the jumps are not straight. The response of the canals is rapid, occurring in one twenty-five thousandths of a second.

In the frog, maintenance of position is evidently muscular. When the canals are present, they keep the head level and prevent the sway which the utricles would otherwise bring about.

DISCUSSION

DR. SAMUEL J. KOPETZKY, New York: Since the compact bone in the mastoid should not be thought of as pathologic, and since egress only is necessary to stop cholesteatoma, why should one do more than remove the diseased portion and afford ample drainage? There is one exception to this: When the cholesteatomatous matrix involves intracranial structures, the whole matrix should, of course, be removed. In otosclerosis the lesion is in unique bone. The lesion is not unique, for it is found in other diseases, such as cretinism, osteomalacia and Recklinghausen's disease. There is a functional disturbance in the nervous system. Three important factors are as follows: 1. There is a "trip" in the calcium fixation somewhere in the body. 2. The bony labyrinthine capsule is the only bone developed fully at birth. Therefore, the time to study otosclerosis is in the intra-uterine period. If it were possible to obtain fetuses from families with a hereditary tendency to otosclerosis, one might find the cause. 3. Deafness is secondary to nerve degeneration after birth.

THE ANATOMY OF THE TYMPANIC MEMBRANE. DR. O. V. BATSON, Philadelphia (by invitation).

Since the clinical approach to the membrana tympani is by way of the external auditory canal, the changes that occur in the canal with age are of interest. In infancy the canal is arched upward in its middle portion. As age advances this curve commonly becomes flattened, so that by middle life the course of the canal approaches the horizontal. This seems to be associated with the flattening of the middle cranial fossa. Casts of the canal made at various ages show that the manipulations used to facilitate examination of the drum membrane are useful only in alining the auricle with the canal and probably produce no change in the canal itself.

Numerous aural microscopes have been developed to make clinical observations. The clumsy and complicated ones defeat their purpose. It should be remembered that the resolving power of the objective and not the magnification of the ocular is significant in a diagnostic instrument. It is then readily seen that the small telescopic magnifiers give as much wealth of detail as the larger instruments now available.

The data obtained from specimens prepared for this study agree with the published accounts of the vascular supply of the drum membrane. The vessels radiate from the handle of the malleus to the margin. The dual connections—the central

vessels and the peripheral plexus—indicate in part the reason for the ready regeneration of the membrane following pathologic or surgical puncture.

The fibrous structure of the membrane was investigated by the method discovered by Dupuytren and elaborated by Langer. A pointed, round instrument when thrust into the skin produces a slit. The direction of the slit depends on the direction of the predominating fibers. This phenomenon was found to hold for the drum membrane, which was for the first time examined by this method. The method is important because it shows the direction of the major portion of the fibers; the slits are larger in the direction of the major fibers. It is known, further, that rapid natural closure follows any parallel incision in the skin with the cleavages, while drainage is secured by making the incision across the direction of the clefts. Two groups of clefts are produced in the drum membrane, circular and radial. The circular clefts are small and fall into a central and a marginal group. The radial clefts are coarse slits; they are at right angles to the handle of the malleus and radiate from its tip. Examination of the membrane by polarized light contributed no additional information.

This study indicates that the usual paracentesis tympani, between the malleus and the posterior margin, separates the membrane so that there is an adequate supply of blood on either side of the cut, and, further, the incision is so placed in relation to the fibers that maximum drainage will be obtained.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION OF OTOLARYNGOLOGY

Dec. 21, 1932

BENJAMIN PARISH, M.D., *Chairman*

PREVENTRICULOSIS (SO-CALLED CARDIOSPASM): DIAGNOSIS AND TREATMENT (MOVING PICTURE DEMONSTRATION). DR. CHEVALIER L. JACKSON.

The film showed briefly the physiology of swallowing and then the pathologic physiology of preventriculosis. "Preventriculosis" is the term preferred by Chevalier Jackson to designate the condition commonly known as "cardiospasm."

The etiology and pathogenesis of this condition have not been entirely determined, but in typical cases of this disease there occurs a functional stenosis at the level of the diaphragm, associated with more or less dilatation of the esophagus above. The diaphragmatic pinch-cock, probably composed chiefly of the crura of the diaphragm, fails to open as it normally should in the deglutitory cycle, though on passage of an esophagoscope or other instrument no more than the usual resistance is met with at this point.

The typical roentgenographic appearances of preventriculosis were then shown. While roentgen examination is of great diagnostic aid, the diagnosis should always be confirmed by subsequent esophagoscopy in order to rule out an organic lesion. The technic of diagnostic esophagoscopy was also demonstrated in the film.

The most satisfactory instrument for therapeutic dilation is, in Dr. Jackson's opinion, a pneumatic dilator of the type used by Mosher, with opaque lines in the rubber bag which permit one to place the instrument accurately under fluoroscopic guidance, as well as to observe the outline of the indentation produced on the bag and the amplitude of dilation obtained. This apparatus and the technic of its use were shown in the film. The rubber tubing is calibrated in order to facilitate the taking of measurements at the first (fluoroscopic) dilation for subsequent use when fluoroscopic guidance is not used; this feature was also demonstrated.

LARYNGOFISSURE FOR EARLY LARYNGEAL CANCER (MOVING PICTURE DEMONSTRATION). DR. GABRIEL TUCKER.

The treatment of cancer of the larynx by laryngofissure necessitates early diagnosis. The film outlined the steps for early diagnosis, general examination,

roentgen examination and repeated mirror observations and study. The diagnosis of cancer should be confirmed in every case by biopsy before either surgical treatment or irradiation is planned. A demonstration of the direct laryngoscopic technic for biopsy was shown. Early cancer of the larynx originates most frequently in the anterior portion of the glottis and is amenable to surgical removal by laryngofissure. Laryngofissure is applicable only to this type of growth. More extensive lesions and lesions in other locations may be treated by a thyrotomy incision with wider removal of tissues. This operation, however, should be termed partial laryngectomy. The term laryngofissure should be restricted to the removal of the tissue within the thyroid cartilage only. The technic of Chevalier Jackson for the treatment of growths on the cord and in the anterior commissure was illustrated by drawings, as was also the technic of St. Clair Thomson. It was suggested that the Thomson technic may be used also for partial laryngectomy because of the wide exposure that it gives inside the larynx. Local anesthesia only is required for laryngofissure. In a series of fifty cases of laryngofissure and partial laryngectomy there was no operative mortality. The management of complications following laryngofissure was discussed. The method of treatment of wound infection and the direct laryngoscopic removal of excessive granulations following laryngofissure were demonstrated. The treatment of recurrences, which occur in less than 15 per cent of the cases of laryngofissure, was demonstrated in a patient on whom laryngofissure, partial laryngectomy and total laryngectomy were performed; there had been no recurrence after the total laryngectomy, one and a half years having elapsed. The following conclusions were offered: 1. Chronic hoarseness is a constant symptom in intrinsic cancer of the larynx. 2. The choice of laryngofissure for surgical treatment in intrinsic cancer of the larynx should depend on the location and extent of the lesion. The cellular characteristics are of secondary importance. 3. Laryngofissure for early intrinsic cancer of the larynx will cure more than 80 per cent of the patients, saving the patient's larynx as well as his life. 4. A more radical operation can still be performed in cases of recurrence following laryngofissure if the patient has the proper follow-up observation.

Operation on Abscess of the Temporosphenoid Lobe (Moving Picture Demonstration). Dr. Walter Roberts.

DISCUSSION

Dr. Henry Dintenfass: This series of operations was successfully performed by Dr. Roberts, whose excellent judgment in the diagnosis and in the timing of the therapeutic procedures brought about the complete recovery of the patient.

At the first operation, when simple mastoidectomy was performed and the cofferdam placed in position, I did not have my camera with me, so we substituted a drawing in the film to show what occurred.

The particularly interesting features in this case, of course, were the diagnosis and method of treatment. The symptoms of slow pulse, photophobia, headache and clouded mentality were merely the manifestations of increased intracranial pressure which are associated with an abscess of the brain in any location. The presence of aphasia, however, was a localizing symptom and indicated definitely that the lesion was temporosphenoid. The superior temporal convolution, or Heschel's convolution, was affected. The aphasia was sensory: The patient could understand and recognize objects but was unable to name them; however, he could repeat the name of an object after he had heard it. This condition is to be differentiated from motor aphasia, in which objects cannot be named. The lesion causing motor aphasia is in Broca's region, the inferior frontal convolution. It was fortunate that the abscess was not in the so-called "silent" area, that is, on the right side of the brain in a right-handed person, in which case the localizing symptom of aphasia would not have been present.

Papilledema was absent. Neither did vomiting occur, notwithstanding the size of the abscess. According to Kopetzky, these are late symptoms of abscesses in

the temporosphenoid region. In cerebellar abscess papilledema is apt to exist earlier in the course of the disease because of the proximity of the pus pocket to the foramina of the fourth ventricle, with the consequent blocking of the ventricle by pressure.

The use of the coffer-dam to seal off the inflammatory process and to prevent meningitis was life-saving. In incising the dura great care was taken not to injure the middle meningeal artery. The incision in the dura was made parallel to the temporal convolutions.

The problem of drainage, one of most importance in the treatment of abscesses of the brain, was taken care of by the rubber tube, the abscess being completely emptied after the first insertion. This would give the impression that some form of abscess capsule existed, notwithstanding the acute type of lesion.

It is also interesting to note that the aphasia cleared up the day following the drainage, but the slow pulse continued for several days after the operation, which demonstrates that the adjustment to normal of the increased cerebral pressure is slow.

THE VARIOUS PHASES AND STEPS IN ONE-STAGE TOTAL LARYNGECTOMY (LANTERN SLIDE DEMONSTRATION). DR. FIELDING O. LEWIS.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. W. P. Wherry, 107 S. 17th St., Omaha.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Cleveland. Time: June 11-15, 1934.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Bronchoscopic Society

President: Dr. W. F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

American Laryngological Association

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. W. V. Mullin, 9204 Euclid Ave., Cleveland.

American Laryngological, Rhinological and Otological Society

President: Dr. J. W. Jervey, 101 Church St., Greenville, S. C.
Secretary: Dr. R. L. Loughran, Bridgewater, Conn.
Place: Charleston, S. C. Time: April 3-5, 1934.

SECTIONS

Eastern.—Secretary: Dr. Vernon L. Bishop, Medical Arts Bldg., Rochester, N. Y.
Southern.—Secretary: Dr. Fletcher D. Woodward, P. O. Box, 1685, University, Va.
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President: Dr. John R. Page, 127 E. 62nd St., New York.
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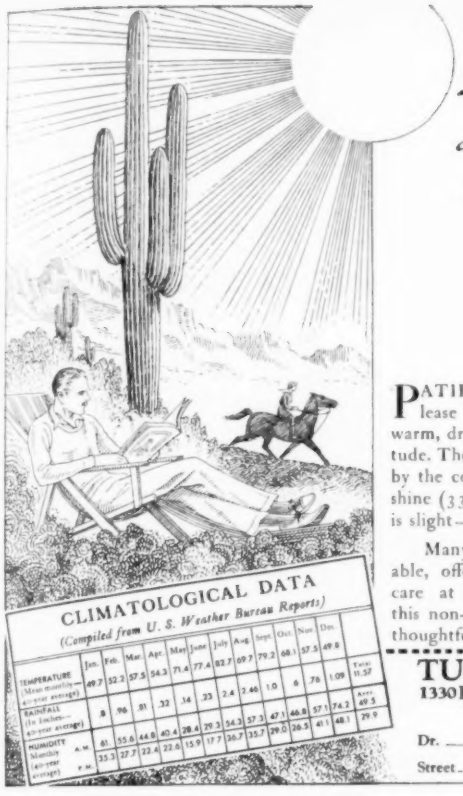
* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.



CLIMATOLOGICAL DATA

(Compiled from U. S. Weather Bureau Reports)

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	
TEMPERATURE (Mean monthly—40-year average)	49.7	52.2	57.5	64.3	71.4	77.4	82.7	80.7	79.2	68.1	57.5	49.8	Ann. 65.57
RAINFALL (In inches—40-year average)	.8	.96	.81	.32	.14	.23	2.4	2.46	1.0	.6	.76	1.09	Ann. 11.11
HUMIDITY Monthly (40-year average) A. M.	61	55.6	44.8	40.4	28.4	29.3	54.3	57.3	47.1	46.8	57.1	74.3	Ann. 49.5
HUMIDITY Monthly (40-year average) P. M.	35.3	27.7	22.4	22.6	15.9	17.7	36.7	35.7	29.5	26.3	40.1	48.1	Ann. 29.9

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Calcium Compounds
Chaulmoogra Derivatives
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Cinchophen and Cinchophen Derivatives
Copper Salts
Creosote and Guaiacol Compounds
Cresol and Cresylic Acid Preparations
Cresol Derivatives
Digestive Enzymes
Digitalis Principles and Preparations
Dyes
Ephedrine
Epinephrine and Epinephrine Preparations
Ergot Preparations
Ethylhydrocupreine
Fibrin Ferments and Thromboplastic Substances
Filicic Acid and Related Substances
Formaldehyde Preparations
Gold Salts
Hydrochloric Acid Substitutes
Hypochlorites and Hypochlorite Derivatives
Insulin
Iodine Compounds
Ipecac Principles and Preparations
Iron and Iron Compounds
Lactic Acid-Producing Organisms
Liver and Stomach Preparations
Magnesium Compounds
Manganese Compounds
Medicinal Foods
Mercury and Mercury Compounds
Naphthol Compounds
Nitrates—Organic
Opium Principles, Derivatives and Preparations
Organs of Animals
Paraffin for Films
Peroxides
Phenetidin Derivatives
Pyrazolon Derivatives
Quinidine
Quinine Derivatives
Radium and Radium Salts
Resorcin Compounds
Rhus Preparations
Salicylic Acid Compounds
Sandalwood Oil Derivatives
Scopolamine
Serums and Vaccines
Silver Preparations
Sulpholchthyolate Preparations and Substitutes
Sulphonmethanes
Tannic Acid Derivatives
Thyroxin
Urethanes (Carbamates), Urea and Ureids
Viosterol
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